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The British Journal of Surgery

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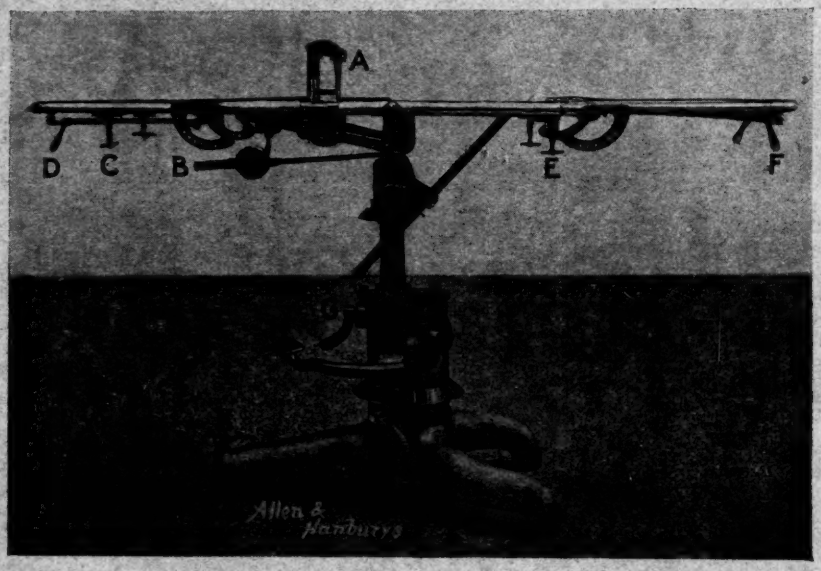

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SIR PHILIP CRAMPTON, Bart.

1777–1858.

The ideals and the outlook on life of the Irish surgeons during the last century were widely different from those of their English and Scotch contemporaries. Dublin, like Edinburgh, was the home of a highly-cultivated and select circle in which members of the medical profession were prominent; in London they had no such advantage. In Dublin the life must have been pleasanter than in Edinburgh; there was less formality; sport and convivial meetings occupied much time; and if the *odium medicum* was as prevalent in Ireland as in Edinburgh, it vented itself in a duel and a reconciliation rather than in a war of pamphlets. It is not surprising, therefore, if the influence of the great Irish surgeons was individual rather than general; if it was the result of personality more than of the writings which they left behind them. Yet they exercised a very real influence on the progress of surgery, an influence which extended from them through their pupils to the surgeons in Great Britain.

Sir Philip Crampton was a member of the band of Irish surgeons who made Dublin famous. He belonged to a family which migrated originally from South Cottingham in Nottinghamshire and settled in Ireland in the reign of King Charles II. Born in Dublin on June 7, 1777, he was the youngest son of John Crampton—who lived in Merrion Square, where he had built up a lucrative dental practice—and Anne, daughter of James Verner. At the age of 14 young Crampton was apprenticed to Solomon Richards, who was four times President of the Royal College of Surgeons in Ireland, and was reputed to be the fattest surgeon in the United Kingdom. He thus began his professional career at the College School in Mercer Street and at the Meath Hospital, to which Richards was attached as surgeon. In 1798—the year of the Irish Rebellion and of the French invasion—Crampton applied for a commission as 'Surgeon's Mate' or Staff Assistant Surgeon, and presented himself for examination for Letters Testimonial of the Irish College. He put in his first day's examination, but during the time which was to elapse between the first and second examination he was ordered to join the army of Sir John Moore, and was present at its encounter with the French at Ballinamuck. During his absence on this service he was appointed surgeon to the Meath Hospital and he thus became surgeon to the hospital before he was legally qualified to practise. On his return to Dublin he passed the second part of the examination, and received the Letters Testimonial of the College on September 25, 1798. He then visited Glasgow, Edinburgh, and London, finally graduating M.D. at the University of Glasgow in 1800.

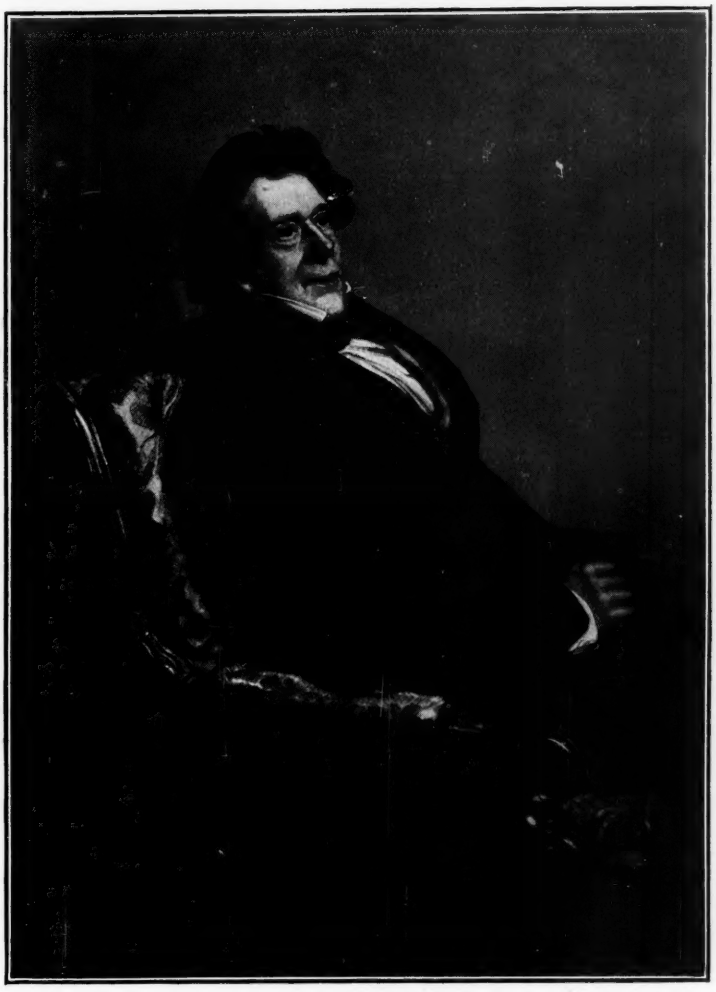
Returning to Dublin, he began to practise in Dame Street, and moved soon afterwards to 24, Dawson Street, being admitted a Member of the Royal College of Surgeons in Ireland on August 11, 1801, and a member of the Court of Assistants in the same year, when he was not yet 25 years of age. In 1804 he fitted up a loft over the stable at the back of his house in Dawson Street as a dissecting room and thus established the first private medical school in Dublin. He lectured on physiology, pathology, and surgery, and associated himself first with James Towell and afterwards with Peter Harkan to teach anatomy. Harkan was well known as a successful resurrectionist, and always headed the parties of students who made forays for subjects. On one occasion, whilst exploring in 'Bully's Acre', the 'watchers of the dead' made a rush at him and his companions. Harkan got all his assistants over the cemetery wall, but, whilst crossing over himself, his legs were captured by the watchers. His pupils seized him on one side and his captors on the other, and subjected his body to a severe see-saw movement, which injured him so severely that he never quite recovered, and died a young man in 1814. The school was so successful that Crampton closed it at the end of the session 1812–13.

Crampton came before the notice of the public in Dublin in 1812, because he happened to live opposite the Richmond Tavern in Dawson Street, where a greedy waiter, whilst removing a dish, attempted to swallow a piece of meat. The meat stuck in his throat, and he fell insensible on the floor. Crampton was hastily summoned, performed a tracheotomy on the spot, and the man recovered. The boldness, skill, and decision of the young surgeon became the universal topic of conversation and a subject of the highest commendation in the public papers.

In 1806 he was appointed a surgeon to the Westmoreland Lock Hospital, and in 1811 he was chosen President of the College of Surgeons. Two years later the Duke of Richmond, then Lord Lieutenant, nominated him to the important and lucrative position of Surgeon General. It is recorded that when he attended his first levée he attracted much notice by his splendid physique and magnificent uniform. Some one asked who he was,

and witty Judge Norbury instantly replied, "He is a General in the Lancers." He resigned his appointment at the Lock Hospital when he became Surgeon General. Early in the reign of King George IV he was appointed Surgeon in Ordinary to the King in Ireland, and he filled the same position during the reign of Queen Victoria. In 1839 he was created a baronet. He was a member of the senate of the Queen's University of Ireland and of the London University

He married Selina, third of the eight daughters of Patrick Hamilton Cannon, an officer of the 8th Dragoons, and by her had two sons and four daughters. He was succeeded in the baronetcy by his son, Sir John Fiennes Crampton, K.C.B., who served as British Ambassador at the Courts of St. Petersburgh and Madrid, and died without issue, the title thus becoming extinct. Sir Philip died at his house in Merrion Square on June 10,



SIR PHILIP CRAMPTON, Bart.

From the engraving by D. Lucas of a painting by W. Stevenson.

1858, and his body, encased in Roman cement as he wished, was buried in Mount Jerome Cemetery.

Crampton was more distinguished as an operating surgeon and a clinical teacher than as a writer. His forte lay in rapid and accurate diagnosis—a look, a touch, one or two pertinent questions, and the diagnosis was made, the treatment determined upon. He seldom erred. To the last his hand was light and steady, his movements during an operation were graceful, devoid of show, rapid without being hurried. He was cool in every emergency and prompt in every danger. His appearance was striking—about six feet in height, slightly built, well proportioned, elastic as corkwood, with large and well-shaped forehead, and hair abundant even in old age. He maintained his vigour until late in life, and one day when well advanced in years he was heard to say that he had swum across Lough Bray, ridden into Dublin, and amputated a limb before breakfast. Throughout his life he was passionately fond of outdoor exercises. He was a first-rate rider, kept his own pack of hounds, and seldom missed a day's sport even when he was at the height of his practice. For eloquence he had no rival in the profession, and his fund of anecdote and raillery was inexhaustible and inoffensive ; yet his temper was so mild that a servant who lived with him three and thirty years said that he never heard a cross word from his lips.

In 1805 Crampton published an *Essay on Entropion or Inversion of the Eyelid*, and in 1813 he described a muscle in the eyes of birds to which the name *musculus cramptonius* was given. The muscle enables the bird to accommodate at will. For this discovery he was elected an F.R.S. He improved the operation for cleft palate, and on March 7, 1834, he removed a stone by lithotrity, being thus the first surgeon to perform the operation in Dublin.

Throughout his life Crampton was interested in animals. He was President of the Zoological Society in Dublin, of which he was one of the principal founders, and was mainly instrumental in obtaining the site in Phœnix Park which is still occupied by the Society.

Crampton's memory was honoured both by the members of his own profession and by the public. His bust in marble, executed by J. R. Kirk, R.H.A., adorns the Royal College of Surgeons in Stephen's Green, and a bronze fountain with a bust of Crampton in front of it is placed at the junction of Great Brunswick Street and College Street.

The cliché of the bust illustrating this article has been kindly lent by the Registrar of the Royal College of Surgeons in Ireland, at the request of Colonel William Taylor, C.B . F.R.C.S.I. The engraving is by D. Lucas from a painting by W. Stevenson. It is dated " Dublin, Dec. 20, 1842 ", and therefore represents Sir Philip Crampton at the age of 65.

SPECIMENS OF LONG BONES SHOWING THE PROCESSES OF INFECTION AND REPAIR:

CONTAINED IN THE ARMY MEDICAL COLLECTION NOW ON EXHIBITION IN THE MUSEUM OF THE ROYAL COLLEGE OF SURGEONS OF ENGLAND.

By Professor ARTHUR KEITH, F.R.S., Conservator of the Museum,
and Captain MORTON E. HALL, C.A.M.C.

In the preceding number of this Journal we described the types of fracture which occurred in long bones when struck by missiles. In this article we deal with the related processes of infection and repair of fractures, so far as they are demonstrated in the specimens in the War Office Collection. It will be recognized that the military surgeon, in

FIG. 272*a*.

FIG. 272*a*. — No. 6.26. Medial surface of the upper third of a right femur, showing the aperture of entrance of a shrapnel ball, and extensive comminution. Note the formation of callus on the fragments, and the goblet-shaped area of necrotic bone in the inferior fragment.

FIG. 272*b*. — No. 6.26. Posterior surface of a bone, showing extensive comminution and the aperture of exit of the missile. Note the callus buds scattered over the several fragments.

FIG. 272*b*.

treating fractures, has had to deal with a problem which is quite different from that which concerns the civil surgeon. The soldier in the fighting zone had to live under conditions which made personal cleanliness impossible. When injured by a projectile, organisms were carried into the tissues and bone by the missile, with pieces of infected clothing and skin. The great majority of injuries were, therefore, infected from the very beginning. Then, not unusually, in the rush of a big push, the casualties were enormous. Men lay out in the open for many hours without treatment. When they arrived at the casualty

clearing station, local sepsis had already begun, and then there was the necessary delay before they reached the base hospital where thorough and continued treatment could be carried out. All these circumstances must be kept in mind when a study is made of the pathological processes exhibited by the specimens in the War Office Collection. Also, comminution is inevitable at the site of a gunshot injury to a bone ; fragments are thrown off in every direction into the tissues. It was difficult to correct deformity under such conditions of infection and comminution.

The War Office Collection contains a large number of specimens which emphasize the difficulties that the surgeon had to contend with. In this article we intend to present a number of specimens to show how the process of repair has progressed, and in what ways

FIG. 273*a*.

FIG. 273*a*. — No. 6.28. Anterior surface of the shaft of a left femur, showing a 'butterfly' type of fracture and the aperture of entrance of a bullet. Note the margin of smooth dead bone around the fragments, with the central area of new callus.

FIG. 273*b*. — No. 6.28. Posterior surface of the same specimen, showing the aperture of exit, with more extensive comminution than that seen about the aperture of entrance.

FIG. 273*b*.

the repair has been modified or inhibited by extensive comminution, and by the infection carried into the site of injury with the missile. The histories of our cases are, in many instances, incomplete or not available ; but where obtainable, as complete a history as possible has been given.

We have classified the specimens presented according to the anatomical area involved —femur, upper, middle, and lower third ; knee-joint ; etc.

INJURIES OF THE UPPER THIRD OF THE FEMUR.

In specimen No 6.26 (*Figs.* 272*a* and 272*b*) there is an extensive fragmentation of the neck and trochanteric region of a right femur. The missile, a shrapnel ball, passed from before backwards, entering lateral to the small trochanter, and made its exit above

the posterior intertrochanteric line between the two trochanters, blowing out a large aperture of exit, and shattering the trochanteric region into several large fragments. The injury was infected, and the process spread to the hip-joint. The fragments of bone apparently retained circulatory connection, as they show evidence of reparative changes. This is not the case with the fractured portion of the femur neck. The evidences of repair consist in fine callus buds scattered irregularly over the fragments, particularly marked on the main inferior fragment. In the latter, the callus is separated from the fracture line by a margin of dead bone, which is not eroded, but is outlined by a trough along which separation is taking place to form a sequestrum. The articular surface of the femur head is not eroded in spite of the infection present. The soldier died from sepsis. No further history could be obtained.

FIG. 274. — 6.3164. Antero-lateral surface of a right femur, showing a spiral fracture with the deposit of callus along the fracture lines produced by seven weeks of attempted repair. The sequestra are seen along the fracture margins, and between the two main fragments a smaller fragment, driven into the medulla at time of injury.

Specimen No. 6.28 (*Figs.* 273*a* and 273*b*) is that of a left femur showing a 'butterfly' type of fracture. The bullet has passed through the shaft from front to back at the junction of the upper and middle thirds. The fracture will be seen to have taken place by the splitting off of a wedge of bone on each side of the aperture of exit. The wedges each show comminution. Although it is not known how long after injury the specimen was obtained, it is evident that considerable reparative changes have taken place. Each of the fragments shows new callus formation on the surface, but along the fracture line of each there is a narrow zone of smooth dead bone, which is separated from the living portion by a groove produced by absorption.

INJURIES OF THE MIDDLE THIRD OF THE FEMUR.

Specimen No. 6.3164 (*Fig.* 274) shows a spiral fracture involving the middle third of the right femur produced by a fragment of high-explosive shell which penetrated the lateral surface of the shaft, driving inwards a portion of the cortex. The fracture has been a fairly simple one without appreciable comminution. The soldier was admitted to hospital with multiple wounds, of which the one of the thigh was the most serious. He was wounded on Oct. 3, 1917, and lived for seven weeks, and the specimen illustrates the failure of union due to sepsis and necrosis. Extensive areas of the fracture surfaces are dead, and are being separated and thrown off as sequestra. Along the fracture line internal to the sequestra, an abundant callus network has been formed. The deposits from the two main fragments have not been able to unite, however, on account of the presence of the sequestra, and non-union has resulted. The piece of in-driven bone (2 × 4 cm.) lies between the two fragments, and is adherent to the upper. It shows marked infective erosion, as do the fracture surfaces of both fragments generally.

INJURIES OF THE LOWER THIRD OF THE FEMUR.

Specimen No. 6.3551 (*Figs.* 275*a* and 275*b*) shows a compound comminuted fracture of the lower fourth of the shaft of a left femur caused by a rifle bullet. The comminution has been extensive, and there has been a loss in continuity of the shaft of several centimetres. The site of injury was infected, and the various fragments show extensive necrosis and partial separation of sequestra. The process of infection has spread into the knee-joint, and the patellar and posterior condylar portions of the articular surfaces show considerable erosion. Along the fracture lines there are areas of smooth dead bone

FIG. 275*a*.

FIG. 275*a*.—No. 6.3551. Anterior surface, lower third, of a left femur, showing a comminuted fracture and the process of repair which has taken place during a period of 43 days. Note the scattering of the fragments, and the areas of smooth dead bone being separated as sequestra; also the lacelike callus deposits.

FIG 275*b*.—No. 6.3551. Posterior surface of the same specimen as in *Fig.* 275*a*.

FIG. 275*b*.

separated from the living portions by a groove. Beyond the line of separation fairly abundant callus has been laid down, but it is of that spongy type ordinarily found where infection is present. The soldier was wounded on April 11, 1917, and an amputation was performed on May 24 on account of suppurative arthritis.

Specimen No. 6.3901 (*Fig.* 276) shows an incomplete fracture in the lower fourth of a right femur, caused by a shell fragment which passed through the outer side of the thigh. The missile apparently just grazed the lateral surface of the bone, and at this point an oval area of cortex 4 × 2·5 cm. is eroded and undergoing necrosis. There is a complex series of fissures, which are filled in with a deposit of new bone; this was piled up in one place several millimetres in thickness. The cortex adjacent to the fracture lines is eroded, and callus points are scattered over it. The knee-joint was opened into at the time of the injury, and an acute suppurative arthritis developed. The greater portion of the articular surfaces is eroded. The soldier was wounded on June 3, 1917, and an amputation was

performed on July 9, because of the severe continuous infection of the wound and the knee-joint.

Specimen No. 6.36 (*Fig.* 277) is a very instructive and valuable specimen, in that

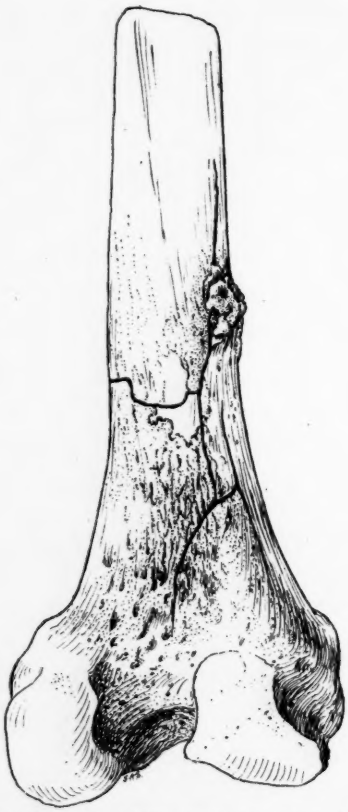


FIG. 276.—No. 6.3901. Posterior surface of a right femur, showing an incomplete fracture, with a complex series of fissures which have been filled in with callus.

FIG. 277.—No. 6.36. Anterior surface of a left femur and knee-joint, showing a compound comminuted fracture of the lower third of the shaft, followed by malposition and necrosis of the broken ends, excessive callus formation, and infective erosion of the knee-joint.

one can trace the complete history of a comminuted gunshot fracture of a femur, the effort which Nature makes to repair the injury, and the difficulties which the surgeon has to face under such conditions, ending with removal of the injured limb by amputation above the site of injury. Several inches of the lower third of a left femur have been shattered into various fragments, and the main lower fragment has been drawn upwards and inwards for some distance, so that it is over-ridden by the superior fragment of the shaft, producing marked deformity; also the two fragments have remained several millimetres apart. Apparently numerous small fragments were removed shortly after the injury was sustained. The soldier was wounded on Oct. 8, 1915, and on Nov. 3, sixty-four days after injury, an amputation was performed in the middle third of the thigh on account of

the extensive deformity, sepsis, and the non-union of the fracture. The ends of the main fragments consist of smooth dead bone and are partially separated as sequestra. These are enmeshed in masses of new bony callus. This callus is excessive, and does not unite the two main fragments. It is formed in irregularly shaped masses, lacelike in character and density, and shows, in places, secondary infective erosion. The knee-joint shows evidences of extensive suppurative changes. The articular surfaces of the condyles, patella, and tibia are eroded, and the patellar surface of the femur is nodular in appearance, due to the proliferation of callus nodules.

INJURIES INVOLVING THE KNEE-JOINT.

Specimen No. 6.372 (*Fig.* 278) shows a gunshot wound of the popliteal area of a left femur, by which the internal condyle has been fractured and detached. It has been displaced in an upward direction, and there has been considerable loss of bony tissue from its medial, posterior aspect. The history of the specimen is not known, but apparently a considerable time intervened before it was removed by amputation, for, in spite of severe infection, the process of repair has been extensive. Just above the superior tip of the condylar fragment there is an area in the upper fragment which is smooth and hard and in process of separation as a sequestrum. Above this area a large mass of callus has been laid down. Similarly, the various condylar fragments are sealed together by callus, but the main fragment has not been so united. The cortex of the condylar fragments and the main shaft, along the fracture line, show necrosis and erosion due to infection. Also the articular surface of the lateral condyle is similarly eroded.

FIG. 278.—No. 6.372. Anterior surface of a left femur, showing fracture and upward displacement of the medial condyle. Note the infective necrosis of the cortex of the fragments, and the large callus mass on the superior fragment.

FIG. 279.—No. 6.51. Left knee-joint, showing extensive disorganization following a gunshot injury. The lower end of the femur shows marked necrosis. Note the irregular callus formation.

Specimen No. 6.51 (*Fig.* 279) shows marked destruction of a left knee-joint by a gunshot, in which the missile passed, from left to right, transversely across the joint, just above the patella. Following the injury there has been disorganization of the joint surfaces, with extensive necrosis of the lower end of the femur due to septic infection. The infection has extended up the shaft for a distance of several centimetres, and the shaft surface of the part involved is everywhere eroded. New lacelike network of callus has been laid down on the surface of the shaft above the area of injury, and also around the margin of the superior end of the tibia. The superior articular surfaces of the tibia are destroyed by erosion. No history of this case is available.

Specimen No. 6.581 (*Fig.* 280) shows destruction of the articular surfaces of a left knee-joint by acute pyogenic infection following a slight gunshot injury of the medial condyle. The knee was evidently kept in a flexed position. The posterior portions of the articular surfaces of both condyles of the femur are uninjured, while the anterior portions, as well as those of the tibia, show marked erosion, to a depth of several millimetres in places. The patellar surface has escaped injury except at the margin. The missile apparently was a small shell fragment which entered the knee-joint and struck the medial condyle, causing an antero-posterior fissure.

FIG. 280.—No. 6.581. A left knee-joint, showing the change produced by acute pyogenic infection following slight gunshot injury.

FIG. 281.—No. 6.591. Anterior surface of the upper ends of a left tibia and fibula, showing fragments which have become united by callus. There is erosion of the articular surfaces.

In specimen No. 6.591 (*Fig.* 281) the posterior margin of the lateral condyle of a left tibia has been carried away by a missile. Running out from the site of injury are numerous fissures. The lateral margin of the condyle and the head of the fibula have been comminuted, and repair has taken place, the fragments being attached by callus, There has been marked infection of the knee and superior tibio-fibular joints, the articular surfaces showing considerable erosion.

Specimen No 6.5904 (*Fig.* 282) shows a compound comminuted fracture of the medial condyle of a right tibia. The missile, a rifle bullet, came from behind and passed

through the head of the tibia, shattering it. Fissures extend down the shaft and across the articular surface. The soldier was wounded on Nov. 27, 1917, and lay out three days before evacuation. He was admitted to base hospital on Dec. 3, and an amputation was performed on account of severe infection of the knee-joint. It will be noted that erosion of the cortex along the fissures had already begun although the period of infection had been brief. The articular surfaces of the tibia had, however, escaped.

In specimen No. 6.6301 (*Fig.* 283) there has been an extensive comminution of the whole upper third of a left tibia by a missile, probably a shell fragment, which perforated the leg from side to side. The soldier was wounded on April 15, 1917, and extension was applied till June 2, when an amputation was performed because of the severe infection, which particularly involved the knee-joint. Partial ankylosis of the latter had occurred, and the soft structures of the joint had degenerated. It will be seen that the entire upper third of the tibia, including the head, has been shattered into numerous fragments, some of which have been lost. Of those remaining, several died at the time of injury, and remain as smooth sequestra, while others survived, and on their surfaces new bone has been thrown out. In some of these, the margins were destroyed and have become entirely separated as sequestra. There has been complete destruction of the articular surface of the head of the tibia.

FIG. 282.—No. 6.5904. Antero-medial surface of a right tibia, showing shattering of the head by a bullet. Note the erosion of the cortex on the surface of the fragments along the fissures.

FIG. 283.—No. 6.6301. Anterior surface of a left tibia, showing the extensive comminution caused by a through-and-through shell-wound. Note the areas of smooth dead bone (sequestra), and the abundant callus on the surface of some of the fragments.

INJURIES OF THE TIBIA AND FIBULA NOT INVOLVING THE KNEE-JOINT.

Specimen No. 6.61 (*Fig.* 284) consists of the upper extremity of a right tibia and fibula removed by re-amputation thirty-four days after injury. The right leg had been nearly blown off by a shell and an amputation performed at once. Infection continued in the stump end, and it became necessary to re-amputate above the knee. The specimen shows three points of interest—namely, necrosis and erosion of the inferior end of both bones, partial separation of sequestra, and massive callus buds above the margin of the sequestra.

FIG. 284.—No. 6.61. Anterior surface of upper extremities of right tibia and fibula, showing necrosis and infective erosion. Note the partial separation of sequestra, and the callus spurs above them.

In specimen No. 6.667 (*Fig.* 285) several inches of the shaft of a right tibia have been comminuted by a bullet. The injury was received on Feb. 24, 1915, and on March 17 an operation for removal of necrotic fragments of bone and drainage of the wound was performed. Two days later secondary hæmorrhage occurred, and amputation above the knee was necessary. The specimen shows a loss in continuity of eight centimetres. Several small fragments remain attached to the upper fragment. Repair has gone on to a considerable extent. Portions of the fragments along the fracture lines are smooth, and are being separated as sequestra from the areas that survived. These latter show an irregular deposit of callus along the line of separation.

FIG. 285.—No. 6 667. Anterior surface of a right tibia and fibula, showing a loss of continuity in the shaft of the former and a separation of sequestra along the fracture lines. Note the new callus formed on the surface of the fragments.

Specimen No. 6.65 (*Fig.* 286) is an excellent example of the changes which take place in the region of a gunshot fracture of bone in the presence of infection. There is an oblique fracture of a right tibia at the junction of the upper and middle thirds, and a similar fracture of the right fibula in its upper third. About each fracture line a similar condition is seen. There is very slight comminution. On each side of the fracture line is a strip of smooth dead bone, varying in width from 3 to 20 mm. The bone adjacent to these smooth dead areas has survived the injury, and has been

markedly eroded by infection for a distance of several centimetres from the line of fracture. Also fine callus buds have developed on the surface. The smooth dead areas have been partially separated by absorption from the portions that survived.

Specimen No. 6.663 (*Fig.* 287) shows a condition of repair and separation of sequestra similar to that seen in No. 6.65 (*Fig.* 286). There is a comminuted fracture of the middle third of the shaft of the left tibia, and a similar fracture of the upper third of the left fibula. In this case the suppuration in the wound was so marked that an amputation above the knee had to be performed. The time elapsing before the amputation was done is not known.

FIG. 286.—No. 6.65. Anterior surface of a right tibia and fibula, showing an oblique fracture of the shaft of each, and the process of repair in the region of the fractures. Note the areas of smooth dead bone being separated off as sequestra.

FIG. 287.—No. 6.663. Anterior surface of a left tibia and fibula, showing comminuted fractures of the shaft of each in which partial repair has taken place. Areas of smooth dead bone are being separated along the fracture lines, and callus buds have formed above and below these areas.

Specimen No. 6.62 (*Fig.* 288) consists of the upper extremities of a left tibia and fibula removed by re-amputation. The specimen illustrates the manner in which bone is being absorbed at the site of a former amputation. The antero-lateral surface of the tibia, extending upwards from the inferior tip to a short distance below the tuberosity,

consists of smooth dead bone, which is seen to be sharply outlined and separated from the portion of the shaft which survived. The line of separation is marked in places by a deep groove where absorption has taken place, while in other parts the line of demarcation is not so distinct. Apertures, representing areas of softening, can be seen here and there in the area of smooth bone. Beyond the line of demarcation, callus has deposited on the bone that survived. The fibula shows a similar separation of a sequestrum at its inferior end. It is probable that the smooth areas of dead bone were destroyed at the time of injury, and, being so extensive and associated with infection, a re-amputation became necessary. No history of this case could be obtained.

FIG. 288.—No. 6.62. Anterior surface of a left tibia and fibula, upper end, removed by re-amputation. Note the areas of smooth dead bone, which were being separated as sequestra.

FIG. 289.—No. 6.666. Anterior surface of a tibia, showing reparative changes following a comminuted fracture. Note the sequestrum partially separated from the living portion of one of the fragments.

Specimen No. 6.666 (*Fig.* 289) shows the reparative changes which have taken place in a tibial shaft following a comminuted fracture caused by a gunshot wound. There are several fragments, of which the superior shows marked necrosis and infective erosion, but no callus formation. The remaining fragments show erosion, as well as reparative changes. They have become united to the lower fragment by new callus, which has also been thrown out profusely on the surface of the fragments. A portion of one of the fragments is smooth and has been partially separated as a sequestrum The soldier was wounded on Aug. 20, 1916, and the specimen was obtained by amputation on Sept. 25.

Specimen No. 6.668 (*Fig.* 290*a* and 290*b*) is remarkable in showing the extensive attempt which Nature has made to effect repair and compensate for a comminuted fracture of both tibia and fibula followed by malposition of the fragments. The injury occurred during the autumn of 1914, and it is supposed that on account of the difficulties of evacuation the patient did not receive the usual and necessary treatment. Some months later the leg was amputated above the site of the deformity. The lower fragment of the tibia, displaced upwards and inwards, has become united to the upper fragment by firm bony callus. The lower fragment of the fibula has become joined to the inferior end of the upper fragment of the tibia. The upper fragment of fibula, separated from the inferior fragment by a distance of several centimetres, has become united to the latter by means of a semi-detached fragment surrounded by new bony callus. In this way

FIG. 290*a*.

FIG. 290*a*.—No. 6.668 Anterior surface of a left tibia and fibula, showing the deformity resulting from a comminuted fracture of both bones in which union has taken place in a peculiar manner. Note the infective erosion of the inferior ends of both bones.

FIG. 290*b*.—No. 6.668. Posterior surface of same specimen as *Fig.* 290*a*.

FIG. 290*b*.

there has been considerable shortening of the leg. The continuity of the tibia has been restored not only by the original inferior fragment of the same bone, but also by the inferior fragment of the fibula. In this case there was also an injury to the ankle-joint which later necessitated an amputation. The inferior ends of both tibia and fibula show necrosis and considerable infective erosion.

Specimen No. 6.71 (*Fig.* 291) consists of the lower two-thirds of a tibia which was removed as a sequestrum from a case of gunshot wound in the upper part of the leg. The crest of the tibia forms a separate fragment. At the upper end of the specimen there is a narrow rim of smooth dead bone which has been unaffected by the infective changes

which have gone on in the remaining portion of the specimen. The latter, including the portion of the tibial crest, shows extensive infective necrosis. The whole surface is pitted with areas where softening has taken place. It is supposed that the fracture was followed by an acute osteomyelitis of the shaft below the injury. No history is available of the duration of the condition.

Specimen No. 6.663 (*Fig.* 292) shows a compound comminuted fracture of the middle third of a left tibia and the upper third of the left fibula. The specimen illustrates the separation of sequestra along the fracture lines, and demonstrates how the presence of dead bone between the normal portions inhibits the formation of normal callus union. It is not known how long after injury the specimen was removed by amputation. In the tibia, the bone along the lines of fracture is smooth, and apparently died at the time the injury was sustained. The areas of smooth bone are separated from the areas which survived by a groove

FIG. 291.

FIG. 291.—No. 6.71. Lower two-thirds of a tibia, showing extensive necrosis and infective erosion, the specimen being removed as a sequestrum at operation.

FIG. 292.—No. 6.663. Anterior surface of a left tibia and fibula, showing separation of sequestra at the site of fracture.

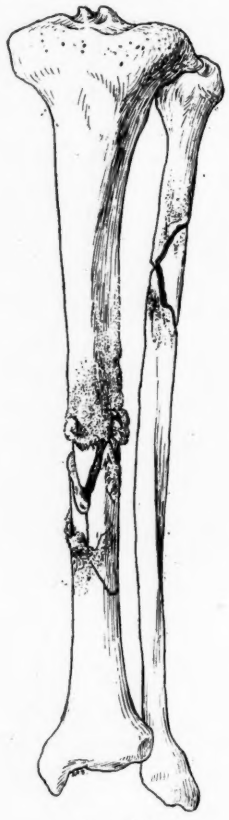


FIG. 292.

formed by softening. Above and below the smooth dead bone, callus has been thrown out on the surface, but there is no attempt at a junction across the fracture line. The cortex of the surviving areas, for some distance from the fracture, shows infective erosion. The fibula shows a condition at the site of fracture similar to that seen in the tibia.

Specimen No. 6.6611 (*Fig.* 293) shows a gunshot wound of the lower third of a right tibia. The missile, a shrapnel ball, perforated the shaft, entering on the antero-lateral surface. An inverted **Y** fracture has been produced, and a large area of the posterior surface of the shaft, at the aperture of exit, has been carried away. The soldier was wounded on May 9, 1915, and, some weeks after, an operation was performed on account

of necrosis and sepsis in the wound. The specimen is a good example showing separation of sequestra along the fracture lines. The bone which survived, on the upper and lower fragments, shows infective erosion and the diffuse formation of new callus. The corresponding surface of the fibula shows a similar erosion and new bone deposit.

In specimen No. 6.662 (*Fig.* 294) a left tibia has been perforated in its lower third by a shell fragment. The missile has split off a wedge of bone on the posterior aspect of

FIG. 293.

FIG. 294.

FIG. 293.—No. 6.6611. Anterior surface of a right tibia and fibula. The aperture of entrance of the missile can be seen. Note the new bone formation, and the areas of smooth dead bone being separated as sequestra.

FIG. 294.—No. 6.662. Anterior surface of a left tibia and fibula, showing fracture of both bones in the lower third, with separation of sequestra. Note the lacelike appearance of the new callus.

the shaft, and at the same time produced a transverse complete fracture of the shaft. The fibula shows a similar fracture just below the level of the tibial injury. In both bones the specimen shows the effects of infection on the cortex, which is somewhat eroded. New diffuse callus masses have formed on the eroded areas. The portion of bone along the fracture lines is smooth and dead and beginning to be separated. It is not known for how long a period the process of repair extended in this case.

INJURIES INVOLVING THE ANKLE-JOINT.

Specimen No. 6.762 (*Fig.* 295) shows severe gunshot injury of the lower third of the shaft of a right tibia, with loss of a large portion of the lateral surface of the shaft. The fibula has a transverse fracture at the same level. The soldier was wounded on June 8, 1917, and an amputation was performed on July 6 on account of sepsis. There has been considerable healing. The remaining fragments of the tibial shaft are sealed together by firm callus, but the lesser fragments and ends of the two main fragments and irregular areas of the fibular shaft show marked infective erosion. The inferior end of the superior fragment is composed of smooth dead bone, and is partially separated off as a sequestrum. Above this the callus is extensive and forms a projecting spur. Two gaping fissures extend across the inferior articular surface of the tibia.

Specimen No. 6.86 (*Fig.* 296) shows a gunshot wound affecting the posterior aspect of an ankle-joint. The lower ends of both tibia and fibula are fissured, the former having lost a considerable area of cortex, and fissures extend across its inferior articular surface. There is also some damage to the posterior aspect of the astragalus. Amputation was performed several days after injury for sepsis. The cortex of the tibia and fibula in the region of the injuries shows erosion and a small amount of new bone formation.

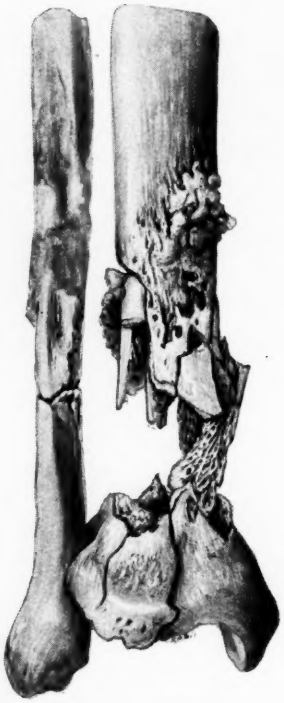


FIG. 295.—No. 6.762. Anterior surface of a right tibia and fibula, showing extensive gunshot injury of the former, with a secondary transverse fracture of the fibula. Note the areas of smooth dead bone and callus spurs.

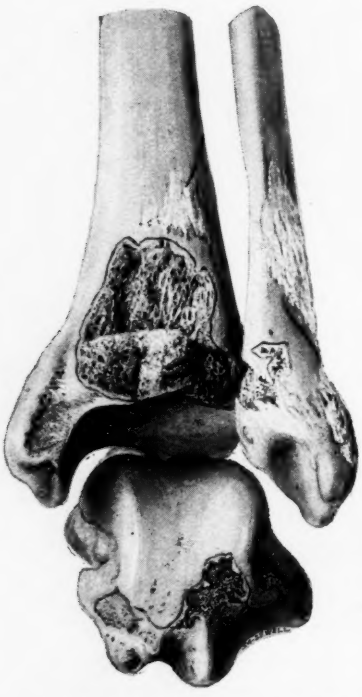


FIG. 296.—No. 6.86. Posterior surface of an ankle-joint, showing a gunshot injury, with erosion of the cortex about the injured areas, due to sepsis.

FIG. 297.—No. 6.802. Medial surface of left ankle and foot, showing severe gunshot injury, followed by infective erosion and new bone formation. The two fragments of tibia are ununited.

In specimen No. 6.802 (*Fig.* 297) there have been marked necrosis and evidence of infection following a gunshot injury of an ankle-joint and foot. Apparently the astragalus

had been excised at an early operation in an attempt to preserve the foot. The lower end of the tibia is splintered, but the fragments remain separate in spite of profuse new bone formation, which is piled up on the surface of one fragment in spur-like processes. There is erosion of the articular surface of the os calcis and its posterior superior surface. The scaphoid and internal cuniform are broken. The history of this case could not be obtained.

GUNSHOT FRACTURES OF THE CLAVICLE SHOWING REPAIR.

Specimen No. 5.133 (*Fig.* 298) shows extensive callus formation with partial union following a perforation of a left clavicle in its middle third. There has been considerable comminution and displacement of the two main fragments. The ends of these fragments consist of smooth dead bone forming sequestra. In spite of the presence of the latter, a large mass of callus has formed on the anterior surface, and has united the two main fragments together. The soldier was wounded on June 7, 1917, and lay in a shell-hole three days before being evacuated. He died on June 19 of empyema and pneumonia, and it is interesting that so much callus formed in such a short interval.

FIG. 298.—No. 5.133. Superior surface of a left clavicle, showing extensive repair following perforation by a shell fragment. Note the dead bone sequestra between the two main fragments.

Specimen No. 5.1335 (*Fig.* 299) shows an ununited fracture, with great shortening, of a left clavicle following a gunshot perforation of the middle third. The soldier was wounded on Aug. 23, 1917, over the left clavicle, and the wound excised the same day. He died Oct. 29 following infection of the pleura, which was penetrated at the time of the original injury. The wound at the site of fracture was very septic. The cortex of the fragments in the region of the fracture shows infective erosion, and abundant new bone has been formed on each fragment, but no union has taken place. Between the ends of the two main fragments a loose fragment of necrotic bone can be seen.

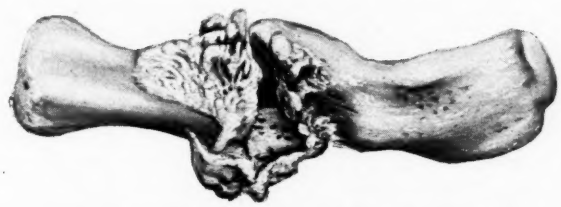


FIG. 299.—No. 5.1335. Superior surface of a left clavicle, showing marked shortening following gunshot fracture. Note the new bone masses, and the necrotic sequestrum between the main fragments.

In specimen No. 5.134 (*Fig.* 300) there has been extensive comminution of the sternal end of a right clavicle, followed by repair extending over twenty-seven days. Some of the fragments have necrosed, and others are involved in a mass of new bony callus thrown out from the ends of the main fragments. No union has taken place. The cortex of the fragments which survived the injury shows infective erosion. The soldier was wounded on April 28, 1917, and died of tetanus on May 25.

FIG. 300.—No. 5.134. Superior surface of a right clavicle, showing extensive comminution of the sternal end. Note the loose dead bone fragments and the new bone formation.

The War Office Collection has recently been enriched by a large number of macerated bone specimens, prepared by Major Fleming at No. 8 Stationary Hospital. Of particular interest in this group are several in which nails or screws have been driven into the tibia for the application of extension apparatus required for the correction of fractures of the femur. In these specimens the screws had been in over a month. A preconceived notion led us to believe that cortical bone tissue tended to react to pressure exerted upon it, and that such foreign bodies tended to loosen within a short period. An examination of this group of specimens would indicate that this is by no means the case. In specimens where loosening of the foreign body has taken place, there is no doubt that the process was brought about by infection. We have come to the conclusion that dense bone will withstand considerable pressure, and that nails and screws driven into bone tend to remain firmly fixed when the conditions of application are thoroughly aseptic.

CONCLUSIONS.

A detailed study of the large number of specimens contained in the War Office Collection showing gunshot fracture of long bones, demonstrates certain points which appear to us important.

In the October number of this Journal we pointed out the types of fracture commonly arising from gunshot wounds The common types were the double- and single-wing 'butterfly' varieties. In many cases the lateral wedges of bone have been shattered into a large number of fragments, and these, at the time of injury, are scattered widely in the tissues about the site of injury. In civil practice, fractures of the long bones, commonly arising from bending or other acts of violence, show no explosive effects. Thus there is seldom comminution, and when this is present, the fragments are not so scattered, nor are they so often separated from their circulatory connection. Union therefore takes place readily, provided the ends of the fragments are in contact. In fractures produced by gunshot, on the other hand, not only is there extensive comminution and scattering of the fragments, but there is extensive tearing and laceration of the surrounding tissues, due to the explosive effects of the missile. In this way certain of the fragments lose their entire circulatory supply, and if infection be present are almost certain to die. These dead fragments represent an insuperable barrier to callus union. Specimens No. 6.3164 and No. 6.6301 (*Figs.* 274, 283), described above, illustrate this point. Callus has formed in spurlike processes on each side of the fracture line, but these have not united with each other.

Another interesting point which we have observed in several specimens in which the healing process has extended over several weeks is that the areas of bone bordering the fracture lines die, and that before union can take place these areas must be removed as sequestra. The margins of dead bone vary from one to several millimetres in width, and it is not unusual to see several fragments in a specimen entirely surrounded by such a rim of smooth dead bone, distinctly outlined from the portions which survived the injury and which show new bone formation. It has been supposed that this destruction of the fracture margin at the time of injury has been due to the stripping off of the periosteum ; but we have come to the conclusion that it is rather due to the shock produced by the missile, which at the same time probably destroys the blood-supply to the areas so affected. Specimens No. 6.6301, 6.667, 6.65, and 6.663 (*Figs.* 283, 285, 286, and 287) illustrate this feature. If shock does not actually kill such areas of bone, it undoubtedly lessens its resistance to infection. If the process of healing has extended over several weeks, the rim appears as smooth dead bone, separated from the surviving portions by a deep narrow gutter produced by liquefaction and osteoclasis.

For some time, in the active-service hospitals, it was the habit to remove all the loose fragments of bone at the site of injury. It would seem to us even more important to consider the removal of the immediate ends of the main fragments, which must otherwise form sequestra, and have to be removed later before union can take place.

Practically all the specimens of fractures of the long bones in which healing has

taken place show evidences of the action of infective agencies. The portions of bone which die at the time of injury remain smooth and unaffected, but those portions that survive show marked changes. It has been noted that the bone becomes considerably lighter in weight, and the cortex, particularly, becomes eroded. In several specimens studied, loose fragments show extensive and diffuse erosion, developing what might be described as a moth-eaten appearance. Infection of a gunshot fracture of a bone need not always be considered as a detriment however. The specimens we have examined show evidence rather that a certain amount of infection present at the site of fracture tends to stimulate new bone formation. In many specimens the cortex between the points of infective erosion has produced numerous and exuberant callus buds. In specimens in the collection where sequestra did not obstruct the process of union, and where death was due to secondary hæmorrhage, or sepsis, the process of union has been widespread, and the callus thrown across the line of fracture has been extensive and effective. This point is of interest in that it offers the surgeon earlier opportunity of undertaking plastic operations, such as plating and bone-grafting, even in the presence of infection, provided there are no other deterrent agents such as the existence of sequestra. It also suggests that it may be important to carry out reparative bone surgery when a certain amount of infective inflammatory change is going on rather than to wait till all evidences of infection have disappeared. The increased circulation and tissue proliferation found at sites of infection can, at the same time, aid in the results of operation.

It must be stated, however, that callus produced in the presence of infection differs from normal bony callus. It is more loosely constructed, and has, in many cases, a lace-like appearance. Specimens Nos. 6.36, 6.372, 6.51, 6.6301, and 6.61 (*Figs.* 277, 278, 279, 283, and 284) show the marked exuberant callus thrown out after several weeks in the presence of infection and sequestra.

We do not wish to enter into a discussion concerning osteogenesis, and as to what part the periosteum plays in new bone formation; but it would seem that even quite dense bone, free from periosteum, will develop large masses of callus, provided its circulatory connections have not been disturbed.

We have been able to distinguish three distinct types of sequestra—namely: (1) Loose dead fragments of bone separated at the time of injury; (2) Loose fragments of bone which apparently survived the injury, but later have become necrotic and extensively eroded on account of severe sepsis; (3) Areas of dead bone along the fracture lines which can only be separated from the surviving portions by operative procedure or the slow process of linear liquefaction. It is apparent that the presence of any one of these sequestra will prevent union in a fracture, and so it is suggested that their removal at an early time after injury offers the best opportunity for an early complete healing.

We wish also to call attention to a point which was noted by Major Rhea, C.A.M.C., that is, the tendency for fine fissure-fractures to extend upwards and downwards along the shaft from a main fracture line. In some cases, when the main fracture is some distance from, and does not involve, a joint cavity, one or more fissure-fractures may extend into the joint. Along such a fissure-fracture, infection may be carried into the joint and set up an acute suppurative process. Several specimens in the War Office Collection show infection of a joint following a gunshot fracture situated some distance from the articulation.

(*To be continued.*)

THE LOSS OF THE ABDOMINAL REFLEXES IN AFFECTIONS OF THE ABDOMEN.

By GWYNNE WILLIAMS, London.

It is difficult to find any mention in the literature of changes in the abdominal reflexes as a result of intra-abdominal disease. It is well known that they disappear in affections of the central nervous system, mainly in those conditions affecting the pyramidal tract, where their disappearance is frequently coincident with the change in the plantar reflex from flexor to extensor. I cannot find any explanation of their disappearance in these conditions, and it is certainly curious that one superficial reflex should disappear while another alters its mode of response.

I have been able to find two papers on the disappearance of the reflexes in abdominal conditions; the first is by Rolleston[1] on their condition in enteric fever, and the following were his conclusions:—

1. The abdominal reflex is affected in a very large number of cases of enteric fever, the percentage of cases in which it is entirely lost exceeding those in which its normal activity is diminished only.

2. From its absence under the age of fifty being confined to certain nervous diseases and acute abdominal conditions, notably appendicitis and enteric fever, the absence of the abdominal reflex in a given case of continued pyrexia in any patient below the age of fifty is of considerable value in diagnosis.

3. The comparatively transient nature of the affection of the abdominal reflex is in striking contrast to the more chronic affection of the knee- and ankle-jerks in diseases associated with peripheral neuritis, e.g., diphtheria.

4. Return of a lost reflex and the resumption of its normal activity are valuable indications of commencing convalescence, and often correspond with lysis and characteristic changes in the fæces and urine.

5. The objective sign of return of the reflex is often associated with the return of the subjective feeling of ticklishness normal to the individual.

6. In re-appearance of pyrexia in convalescence, the condition of the reflex is a valuable index of the nature of the pyrexia.

7. No constant relation exists between the condition of the abdominal reflexes and that of the tendon reflexes.

8. The frequency, degree, and duration of the impairment of the abdominal reflex are, as a rule, in direct proportion to the age of the patient.

The suggestion of these conclusions is that the disappearance of the reflexes is due to toxic causes, though not quite clearly a definite neuritis.

Jamin,[2] in the same year, describes their local disappearance over the areas affected by appendicitis and cholecystitis, and ascribes this disappearance to the influence of the sympathetic on the spinal reflex centres.

Before considering the state of the reflex, it may be well to consider briefly what other changes in the neuromuscular mechanism of the abdominal wall have been recognized.

Rigidity.—It is well known that, apparently as a measure of protection, the abdominal wall can become rigid over an area that is inflamed; for instance, in appendicitis, rigidity may appear over the right iliac fossa while the rest of the abdominal wall remains relatively lax; it may be assumed for the present that this is due to pain, possibly from inflammation of the underlying parietal peritoneum, and that the rigidity is an attempt to prevent movement of the part of the abdominal wall overlying the inflamed part.

Hyperæsthesia.—The change in the sensory condition of the skin of the abdominal wall is not quite so clearly defined, or perhaps it may be said that it is more difficult to estimate. Head drew attention to hyperæsthesia of the area of skin corresponding to the segmental position of the organ affected. That hyperæsthesia over certain areas does occur may be allowed, though the explanation of the phenomenon as evidenced by abdominal disease is not quite satisfactory. In appendicitis, Head stated that there was hyperæsthesia over an area of the anterior abdominal wall lying somewhere between the umbilicus and the right anterior superior iliac spine in cases in which the serous covering of the appendix was stretched, and that this hyperæsthesia disappeared when the tension was relaxed by perforation of the appendix. This phenomenon is certainly not of much value in the diagnosis of the condition of the appendix, but one case may be mentioned which showed it :—

The patient was a woman who had attacks of abdominal pain which came on quite suddenly, lasted a few hours, and then gradually died away ; these attacks had no relation to food, and there was some question as to their cause. When seen immediately after an attack, she had an area of hyperæsthesia in the position described by Head. Operation showed that she had an appendix partially distended beyond a kink ; and there was little doubt that the attacks were due to an acute distention of the appendix brought about by some temporary complete occlusion of its lumen at the kink.

It is difficult to see why the appendix, which is developmentally a middle-line structure, should give rise to a phenomenon situated on the right side, if this hyperæsthesia is to be associated with its segmental position ; and it seems possible that it may really be a phenomenon associated with its present position in the body rather than with its past history, and that it is a local phenomenon of the same class as the rigidity. Unfortunately in this case there is no record of the state of the abdominal reflexes.

It seemed that the abdominal reflexes might throw some further light on these phenomena. These reflexes may be regarded as being four in number—an upper and lower on each side. They are best elicited by lightly scratching the skin with a blunt pin or with a pencil. In young adults they are almost invariably present under normal circumstances, but in old people and those with lax abdominal walls they are frequently difficult to elicit.

The first case to be mentioned is one of appendicitis :—

A woman, age 20, who had been ill for twenty-four hours. The right lower reflex was absent ; there was no rigidity. On operation the appendix was kinked half way along its length, and gangrenous beyond the kink.

It seemed that the absent reflex might indicate inflammation of the area deep to it, and that this absence might be of value in localizing intraperitoneal inflammation. But this idea was soon destroyed, as shown by the next case :—

A boy, age 11, with forty-eight hours' history. He was blue, the abdomen was distended, all the reflexes were present, the muscles were rigid all over the abdomen, and on operation there was pus free in the peritoneum, with appendix in a gangrenous condition behind the terminal portion of the ileum.

The next type of case noticed was that in which an appendix abscess, well defined and therefore some days old, was associated with the presence of all the reflexes. These cases served finally to dispose of the idea that the state of the reflexes was dependent on underlying inflammation.

The following case was very striking :—

A woman, age 25, who was seized with acute abdominal pain twelve hours before I saw her. On admission, the house surgeon found her in great pain and the abdomen rigid all over, and she looked very ill. Three-quarters of an hour after, she appeared quite comfortable and in no pain, in spite of the fact that no morphia had been given. It seemed difficult to believe the previous statement as to her condition. On examination, all her abdominal reflexes were absent, and there was no rigidity beyond some slight resistance in the right iliac fossa. Operation showed free turbid fluid in the peritoneal cavity, with an acutely swollen and inflamed appendix.

This case suggested that the absence of the reflexes was in some way or other dependent on rigidity, and that it might be a phenomenon of fatigue; and the next question was whether it was associated from the beginning with the rigidity, or merely a sequel to this condition. In this connection attention must be drawn to a case of perforated duodenal ulcer seen within two hours of the perforation.

A boy, age 17, who had been seized with acute abdominal pain two hours before admission. When seen he was in considerable pain, and the abdomen was absolutely rigid, but all the reflexes were present. Operation showed fluid in the peritoneal cavity, and a perforated duodenal ulcer.

So far it seems that the disappearance of the reflexes is an indication of a previous rigidity—and, if this be the case, any condition leading to rigidity would give rise to disappearance of the reflex—and that it could not be a phenomenon associated only with affections of the peritoneum. The examination of renal colic demonstrated this to be the case, since it showed that as a rule the upper and lower reflexes were lost on the side of the affected kidney.

One case of renal colic, due to a small stone in the renal pelvis, showed both rigidity and loss of the reflexes after twenty-four hours of pain.

Reappearance of the Reflexes.—A few observations on cases of appendicitis which have been operated upon show that the reflexes reappear in from twelve to twenty-four hours after the operation, provided that the case goes well, and in this respect the following case must be mentioned:—

Male, age 30, operated upon for perforated duodenal ulcer twelve hours after onset. The day after the operation all the reflexes reappeared, but on the second day the lower reflexes again disappeared, and the patient died on the fourth day from general peritonitis, which the post-mortem examination suggested had resulted from a spread from the pelvis rather than from the upper abdomen.

The general indication from these observations seems to be that, as a result of some process within the abdominal area, a process that is usually associated with pain, the abdominal reflexes may disappear for a period of at least twenty-four hours, and that, generally speaking, the reflexes disappear over that part of the abdominal wall which lies over the affected organ. The process producing the change is not necessarily one of inflammation, and not necessarily associated with peritoneum-covered organs, e.g., the reflex disappears in renal colic.

The only change of the parietes which seems to be connected with it is rigidity of the abdominal muscles; but these changes are not completely coincident, the loss of the reflex being, as a rule, later in its incidence than the rigidity.

There are two possible explanations which may be considered, inhibition and fatigue of the reflex; since there is no reason to suppose that the path of the reflex is blocked by any organic change. With regard to inhibition, it might be conceived that it was an attempt to prevent a reflex contraction of the abdominal wall over the painful or diseased part, though it is difficult to see why, if this be the case, the loss of the reflex should not be absolutely coincident with the rigidity, which itself may also be supposed to be protective. Turning to the fatigue of the reflex, the difficulty of finding anything analogous to the loss of a reflex for such a prolonged period is met with. The fatigue of the spinal reflexes investigated by Sherrington[3] showed very short periods, and it was difficult to increase this period by very prolonged stimulation of the reflex. The abdominal reflexes are not purely spinal-cord reflexes; they are lost in lesions affecting the pyramidal tract without loss of the voluntary power over the abdominal muscles; hence it is clear that the reflex path is dependent on a connection with the cerebrum. It is possible that the fatigue of a reflex involving the cerebrum may not show the same laws as those involving the spinal cord alone, and that such a reflex may show a longer period of fatigue than a purely spinal reflex. The difficulty is that it cannot be known whether the stimulus is still present or not, unless the presence of pain is to be regarded as the stimulus. If it is, the fatigue of the reflex certainly lasts some time after the disappearance of the pain.

No relation between the intensity of the pain and the loss of the reflex could be found in the cases observed ; but when the difficulty of measuring pain is taken into account, it is improbable that any such relation would be found.

The question as to where, on the path of the reflex, the fatigue takes place cannot be settled until the path has been defined, and investigations on this point are certainly needed.

CONCLUSIONS.

1. The loss of the abdominal reflexes in abdominal conditions may be regarded as a phenomenon of fatigue of the reflex, this fatigue being due to a previous stimulation of the reflex by a painful affection in the abdominal area ; and it is suggested that the fatigue of the reflex may be cerebral rather than spinal.

2. The local loss of the reflex, e.g., the right lower reflex in appendicitis, or the general loss of all four reflexes in a patient whose abdominal wall is in good condition, is of considerable clinical value ; but its value is not absolute, and it must be determined together with the other features which go to make up the picture of the case.

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[2] JAMIN, *Neurologisches Centralblatt*, 1906, 678.
[3] SHERRINGTON, *Integrative Action of the Nervous System*, 219.

NOTE ON THE 'MONOBLOC' OPERATIVE TREATMENT OF TUBERCULOUS LYMPHANGITIS, WITH AN ILLUSTRATIVE CASE.*

By W. SAMPSON HANDLEY, London.

Though the treatment of tuberculous lymphangitis by the excision of the primary focus, the affected vessels, and the glands of which they are afferents, is recognized as perhaps the only effective treatment for tuberculous lymphangitis of the limbs, it may be doubted whether the importance of excising all the affected tissues in one piece has been properly emphasized. Yet it is just as important to do a systematic 'monobloc' removal of all the affected tissues as in the closely analogous case of a carcinoma, a disease in which the principle is universally admitted. The only difficulties are minor ones—the length of the skin incision, the extent of the dissection—which should include a strip of deep fascia an inch wide along the track of the lymphatic cords—and the consequent tediousness of the operation. Obviously, if the removal is carried out in sections, the chances of a reinfection of the wound are greatly increased.

In the following case the result was not perfect, and the policy of attacking the recurrences, which sometimes gives such satisfactory results in carcinoma, was followed with a considerable degree of success. When last seen, about October 1919, the patient was well.

Case.—**Tuberculous lymphangitis following injury to a tuberculous wart of long standing ; complete excision in continuity of primary focus, infected vessels, and glands.**

E. M., age 47, a piano-maker, for many years had had two small warts on the knuckle of the left index finger. A sister had died of tuberculosis. In April, 1916, he accidentally cut one of the warts with a saw. Slight inflammation followed, and a few weeks later he noticed a small firm swelling under the skin, situated on the dorsal aspect of the lower end of the radius. In January, 1917, he attended Dr. Pringle's out-patient clinic. Tuberculosis of the skin was diagnosed, and he received *x*-ray treatment, but further swellings appeared up the arm, and Dr. Pringle advised surgical treatment. He was admitted under my care on April 20, 1917.

On Admission.—A patch of dry warty lupus, not ulcerated, and about an inch in diameter, was present on the knuckle of the left index. In a line between this lesion and the axilla were numerous nodular swellings (*see Figs.* 301, 302). On the flexor surface of the forearm were several small subcutaneous nodules, covered by normal skin to which they were not adherent. At the junction of the middle and upper thirds of the forearm was a larger fluctuant swelling, ½ in. in diameter, covered by adherent, reddened, and thinned skin. Just above the elbow, in the situation of the bicipital gland, was another swelling, also softening. Several small nodules were present along the inner side of the upper arm, connected by vague cord-like subcutaneous thickenings. The whole formed a chain of nodules from the primary lesion to the axillary glands, which were much enlarged, hard, and not tender.

Operation, April 26, 1917.—An incision was made in the axilla, and the enlarged glands were dissected out without division of the trunks reaching them from below. The incision was then prolonged downwards along the whole length of the arm to the primary lesion, bifurcating where necessary to encircle the larger nodules and the patch of lupus. The nodules, the connecting lymphatic channels, and the primary focus were then dissected out in one strip, continuous above with the axillary glands. Included in the strip were the infected areas of adherent skin over the larger nodules. The wound, which measured 25 in., was then sutured. It healed by primary union. The patient returned to work as a munitioner.

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* The description of this case, but without the coloured drawing, was published in the *Proceedings of the Royal Society of Medicine*, London.

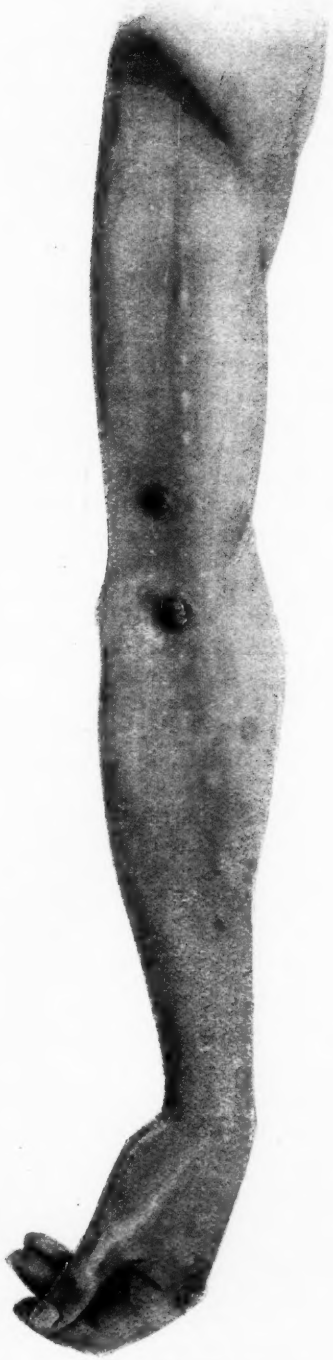

FIG. 301.—Tuberculous lymphangitis of the arm

FIG. 302.—The skin incision.

From a drawing by Mr. W. Thornton Shiells.

On June 2, 1917, he was re-admitted with a small abscess over the middle of the biceps on the inner side of the arm. It was completely excised, together with the infected skin over it. The wound healed by first intention, and he left the hospital on June 11.

In April, 1918, he noticed a warty growth at the side of the original lesion. He was treated by ointments, but the condition grew worse. He continued at work, but the warty patch spread, and in April, 1919, he came again to see me. He had a patch of dry warty lupus, 2 in. in diameter, at the site of the original lesion. One or two subcutaneous nodules, not adherent to the skin, could be felt on the front of the forearm. The lupoid patch was excised, and the area grafted from the thigh. The nodules in the forearm were excised. His general health has continued excellent throughout.

On section, the recurrent lupoid patch presented warty hypertrophy of the skin, with a tendency to downgrowth of the epithelial-cell columns. The papillary region and the superficial layer of the dermis presented diffuse round-celled infiltration, and numerous dilated lymphatic and blood-vessels were seen. The lymphatic vessels showed in places partial obliteration of the lumen by proliferation of the endothelium. A few typical giant-cell systems were present.

Relation of Tuberculous Lymphangitis to Ordinary Lupus.—Cases such as the foregoing raise important questions as to their relationship to ordinary lupus. I have lately become convinced from the microscopic examination of many specimens that lupus vulgaris is essentially and primarily itself a lymphangitis of the cutaneous and subcutaneous lymphatics, and that it differs from 'tuberculous lymphangitis' only in its slow and limited spread through the lymphatics. A close analogy for these extreme variations in rate and extent of spread may be found in acute streptococcal infections of the finger. Some of these give rise only to local redness and swelling, while in others red lines up the arm indicate the spread to the trunk lymphatics of what was primarily a local cutaneous and subcutaneous lymphangitis.

The principal reasons for my belief that lupus vulgaris is a lymphangitis from the beginning may thus be briefly stated. The plane primarily affected, namely, the superficial layer of the dermis, is the plane of origin of the lymphatic vessels of the skin. Changes in the lymphatic vessels of origin can be seen beyond the clinical edge of the area of lupus. Moreover, isolated giant-cell systems can often be found in the subcutaneous tissue, showing that this layer also is affected as well as the skin, and these isolated giant-cell systems are usually found close to the blood-vessels, in the situation of the comitant lymphatics.

If lupus is not a skin affection but a lymphatic disease, its resistance to present methods of treatment can be no matter of surprise. The failure of therapeutic measures based upon false pathology is a constantly recurring theme in the history of medicine. In a subsequent paper I hope to set forth in detail the reasons for thinking that tuberculous lupus is a local tuberculous lymphangitis.

THE END-RESULTS IN PARTIAL AMPUTATIONS OF THE FOOT.

By S. T. IRWIN, Belfast.

During my work at the Limbless Department of the U.V.F. Hospital, Belfast, I have been particularly struck by the difficulties in fitting cases of partial amputation of the foot; by the apparent impossibility of giving such patients even a moderately useful appliance; and also by their frequent return visits for repairs and readjustments of the fitment. My remarks on the subject will be divided under four heads :—(1) *Amputations of the toes*; (2) *Amputations in the foot proper, with special reference to Lisfranc's and Chopart's amputations*; (3) *Amputations in the vicinity of the ankle, with special reference to Syme's amputation*; (4) *Capacity for work in these amputations.*

1. **Amputations of the Toes.**—Trèves, of Paris, makes the striking statement: "The surgeon who retains a painful toe performs a mutilation more grave than an amputation of the leg." In regard to the four outer toes this may be true, provided the painful condition cannot otherwise be relieved; but where relief can be given and the toe retained, as, for example, in hammer-toe by excision of the joint, it is better surgery to retain the toe, and it is always advisable, in my opinion, to retain the great toe if possible. It is rare to find a serious injury or amputation of the great toe which allows a soldier to belong to category A. Even the operations for hallux valgus and hallux rigidus, though they give excellent results in civil practice, were disappointing when done for the serving soldier. A tender scar over the metatarsal head, in amputation of the great toe, was a frequent cause of spasmodic inversion of the foot, and hence produced prolonged disability.

2. **Amputations in the Foot.**—Of all amputations in the lower limb, none give more disappointing results, in regard to function, than partial amputations of the foot. At first they look well; they heal rapidly; the scar is well placed; there are no nerve bulbs; a comfortable limb may even be fitted, the gait may be comparatively good, and the patient may be discharged from hospital with every hope of becoming an efficient worker. Soon, however, he returns, complains of pain, is unable to walk with comfort, can undertake no work; indeed, so much a cripple is he that he is forced to take to crutches again. The frequency of this result forces one to the conclusion that this operation is a failure, only suitable as a primary amputation where conservatism is of first importance, and should never be done as a definitive operation where limb-fitting and suitability for employment are the determining factors in the choice of operation.

The main objection to all partial amputations of the foot is that the anterior pillar of the anteroposterior arch of the foot is shortened. The first effect of this is that the weight acting downwards through the centre of the ankle-joint, and the support acting upwards through the posterior pillar of the arch (the heel), produce a couple allowing rotation round a transverse horizontal axis lying behind the ankle-joint. Therefore the heel moves upwards, owing as well to the upward thrust of the supporting surface as to the pull of the tendo Achillis. Thus a potential equinus is produced, and the long axis of the os calcis comes to be horizontal, or even higher behind than in front. This deformity is best seen in Chopart's amputation, where it is not distorted by the action of any muscles which are inserted into the anterior pillar of the arch of the foot. In those amputations, however, which preserve the insertions of tibials or peronei, the deformity is not a true equinus, but these muscles endeavour to counteract the tendency to deformity, (*a*) by holding up the arch as they normally do, and (*b*) by rotating the foot inwards at the mid-tarsal joint, and so bringing the cuboid as nearly as possible underneath the ankle-joint. Thus a well-marked varus due to muscular contraction complicates

the equinus produced by the couple-like action of weight and support. In many cases this muscular contraction becomes spastic, and the well-known spasmodic inversion occurs.

Tuffier, Depage, Broca, Ducroquet, and Trèves, in various communications, in discussing the relative value of amputations in the lower limb, have shown that the more the anterior limb of the anteroposterior arch is shortened, the worse becomes the result. Disablement is thus increased as we pass from the toes backwards, and the greatest disablement follows an amputation through Chopart's joint. Broca thinks a Lisfranc inferior to an amputation through the metatarsals, a Chopart inferior to an amputation through the scaphocuneiform joint, and both the last two inferior to a Lisfranc.

In regard to this view it is interesting to quote Sir Wm. Ferguson, who in the first edition of his book *Practical Surgery*, published in 1842, wrote : " I would not, however, on all occasions limit myself to the articulations. If, for example, the proximal ends of the metatarsal bones could be saved I would assuredly do so. . . . If, further back on the foot, the cuboid and the scaphoid bones could be preserved, I should do so . . . or divide these bones in their middle with a saw."

In addition to the above-mentioned objections of the shortened anterior portion of the arch and loss of muscular equilibrium, and the deformities of equinus and varus so produced, we find that in these amputations the scar, at first placed anterior or even supero-anterior, comes progressively lower and lower till eventually it lies on the ground ; and last of all, we are faced with the difficulty in supplying suitable appliances for amputation in the foot. Both in France and in Germany elaborate fittings have been invented to restore, by means of springs and levers, some degree of elasticity to the gait. So far as I have heard or seen, these have been uniformly unsuccessful. Depage, commenting on partial amputations of the foot, remarks that whilst the operation results are good, no perfect prosthesis is yet available.

Lisfranc's Operation.—This consists of a disarticulation through the tarso-metatarsal joint, covered by long plantar and short dorsal flaps. Thus the scar lies, at first at all events, over the upper part of the bone, and the covering, supplied chiefly from the plantar skin is well-nourished and in consequence heals rapidly.

Fig. 303.—Photograph of Lisfranc amputation. Showing position of varus.

Symptoms.—These at first are nil, but soon after weight-bearing is allowed, the patient begins to complain of pain in the ankle-joint. This is due to the dropping of the arch and the alteration in articular relationships so produced. To prevent this, contraction of the tibial and peroneal muscles takes place, and pain is referred along them. Soon these muscles become spastic, and a condition indistinguishable from spasmodic inversion occurs. The whole foot now becomes rigid, walking painful, and the patient soon finds himself hardly able to support his own weight on the limb.

Signs.—On examination of the foot, we find that the whole weight is borne on the outer border of the foot, and that the inner edge is held up mainly by the action of the tibialis anticus attached to the internal cuneiform—the tendon of this muscle standing out as a firm rigid cord (*Fig.* 303). This may also be verified by taking a foot-print (*Fig.* 304) with the patient standing, and by taking a stereoscopic radiogram from front to back of the stump. The latter also shows that the cuboid is drawn inwards under the ankle-joint.

Examination of a side-to-side *x*-ray photograph (*Fig.* 305) shows the heel with os calcis drawn up behind, and the cuboid lying beneath the anterior part of the ankle-joint. In an old-standing case a bunion will be found lying over the cuboid, just as one is found in extreme cases of untreated congenital club-foot.

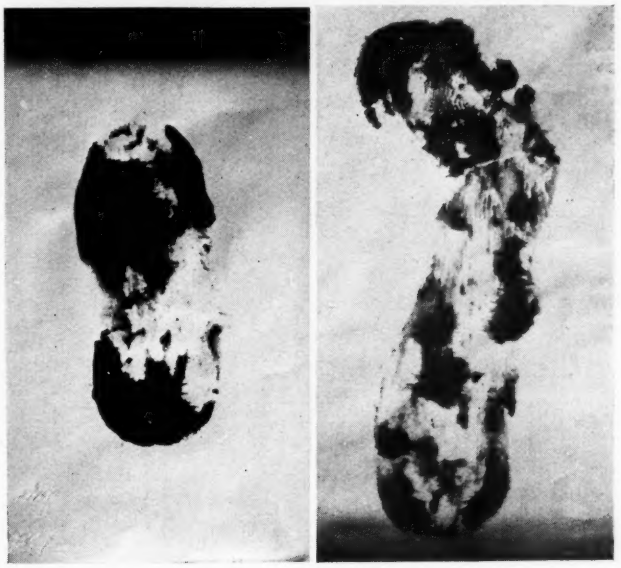

FIG. 304.—Photograph of foot-prints in a case of Lisfranc's operation.

CHOPART'S AMPUTATION.—Briefly put, this is an amputation through the mid-tarsal joint, with long plantar and short dorsal flaps used to cover the bone ends. Just as in the Lisfranc, no fault can be found with the immediate result of the operation. Healing is rapid, the flaps are well nourished, and pressure over the stump end is painless.

Symptoms.—It is only when the foot is used for standing or walking that pain is complained of. This is first of all in the ankle-joint. In this case, however, we have no anterior muscular attachments, and rotation downwards of the front of the os calcis and head of the astragalus takes place, carrying with it the scar, which eventually becomes the bearing surface.

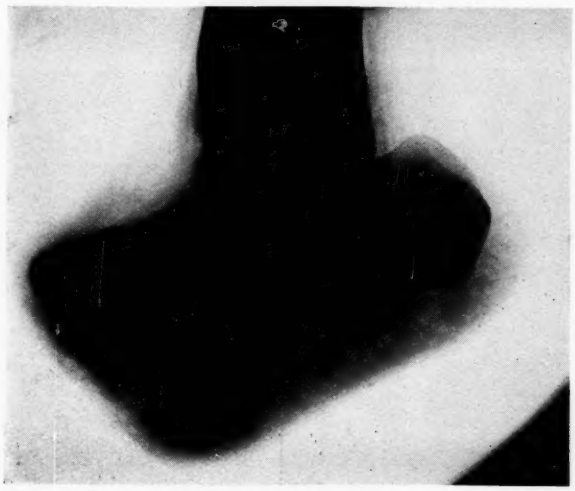

FIG. 305.—Radiogram of Lisfranc amputation. Showing equinus position of os calcis and relation of cuboid to bearing surface of foot.

Signs.—On examination of the stump, we find the main bearing directly under the ankle-joint, the heel being drawn up behind (*Fig.* 306).

A side-to-side *x*-ray photograph of a case of some standing shows the os calcis rotated upwards behind the ankle-joint with its long axis lying higher behind than in front. The scar will be found to have followed the rotation of the bones, and will eventually become part of the bearing surface. There is also a slight degree of varus usually, but this is not nearly so marked as in a Lisfranc.

Various modifications of the amputations of Lisfranc and Chopart have been suggested. The object of the variation has always been to get over some difficulty in the original technique of the operation, and not to secure improvement in function. The latter may be attained to some extent by lengthening as much as possible the anterior pillar of the arch. As Trèves and Broca point out, a Chopart is improved by retention of the scaphoid (*Fig.* 307), a Lisfranc by retaining an inch of the metatarsals. In each case the anterior *point d'appui* is lengthened.

The deformities and disabilities secondary to amputations in the foot may, to some extent, be combated by division of the tendo Achillis, by suture of the extensor tendons to the plantar fascia, and by producing an ankylosis of the ankle-joint. None of these

FIG. 306.—Side-to-side radiogram of Chopart's amputation, showing extreme equinus position of os calcis and astragalus.

FIG. 307.—Modified Chopart (scaphoid retained) amputation. Radiogram showing position of equinus.

artifices, nor all of them combined, produce satisfactory results, and undoubtedly the best course to adopt is to re-amputate, and convert a partial into a complete amputation

FIG. 308.—Radiogram of Syme's amputation. Showing deposit of bone in flap.

FIG. 309.—Stump after Syme's amputation.

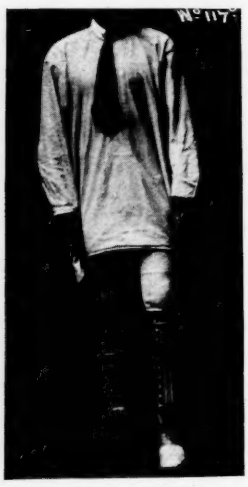

FIG. 310.—Syme's amputation. Stump fitted with artificial limb.

of the foot. The amputations of Chopart and Lisfranc had their place in the days before anæsthetics and antiseptics, when rapidity of operating, and joint surfaces rather than

sawn bone, were important desiderata; but as definitive operations they should not now be performed.

3. **Amputations at the Ankle.**—Of the operations *through the ankle-joint*, that designed by James Syme, of Edinburgh, is *facile princeps* (*Figs.* 308–310). It possesses all the

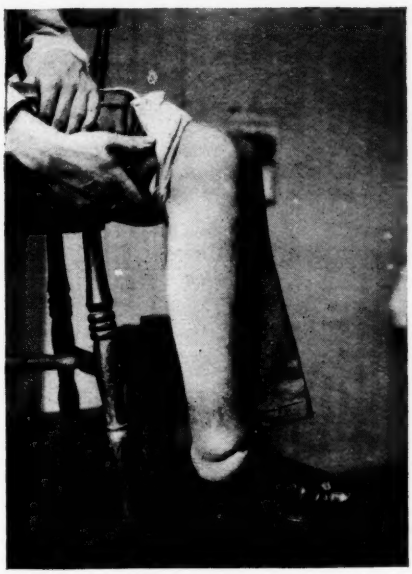

FIG. 311.—Stump after Pirogoff's amputation.

FIG. 312.—Radiogram of stump after Pirogoff, showing non-union of bones.

qualities required for an amputation in the lower limb. The skin of the heel is utilized to take the bearing; it is, therefore, usually complete and permanent. There is ample length of lever, giving firm stability, and this, with the preservation of the natural knee-joint, provide almost, if not indeed quite, perfect gait. The only criticism to be offered is that the artificial ankle, owing to the presence of the side bars, is bulky and rather ungainly. The osteoplastic operation of Pirogoff (*Figs.* 311–313), who saws off the anterior part of the os calcis, retains the posterior part in the heel flap, and brings it up against the sawn surfaces of the ankle bones, at first sight would seem to be superior to the Syme. But in practice there are certain objections: (*a*) Sometimes there is failure to obtain good bony union; (*b*) The skin on the bearing portion of the stump is from the posterior aspect of the heel, and is therefore not true plantar skin; (*c*) The stump so formed is too long, longer actually than the sound side, and in fitting requires an elevation on the sound side to allow of comfortable equable progression. The modifications of Pirogoff, therefore, as well as the other amputations at this level, such as Guyon's, and the subastragaloid disarticulation of Farabœuf, call for no special mention. The fact that the

FIG. 313.—Re-amputation after failure of Pirogoff's amputation.

last retains the astragalus and a functionless ankle-joint, gives it no material advantage; whilst Guyon makes the mistake of sacrificing the plantar skin of the heel, and covering his stump with skin from the posterior aspect of the ankle-joint. They are all improvements upon the partial amputations of the foot, but inferior to the Syme. From the point of view of the limb-maker again, Syme's amputation possesses several important advantages: (*a*) Its shortness allows of the intervention, if required, of a pad over the heel of the artificial limb; (*b*) The plantar skin gives a complete and permanent end-bearing; (*c*) The length of the leg provides ample leverage for activating the limb, and gives the patient that control which is essential to easy natural progression.

Syme's operation is, however, unsuitable (*a*) where injury or sepsis has involved the skin of the heel or the lower ends of the leg bones, (*b*) where atrophy of the skin of the heel has resulted from frost-bite or injury to nerves or blood-vessels. Provided the bone and skin are sound, it is not necessary to get a perfect surgical result in order to obtain good function. The accompanying double Syme (*Fig.* 314) cannot be regarded as above criticism from the point of view of shape, and yet it gave the *amputé* complete end-bearing and an excellent gait.

FIG. 314.—The double Syme's amputation.

4. **Capacity for Work following the foregoing Amputations.**—We now come to consider the results of amputation in the foot from the point of view of function. I have prepared a graph to show how this varies in the amputations of the lower limb. The curve so drawn does not move in a regular manner as we pass upwards, though it will of course be conceded that in general there is a fall in capacity for work as the stump becomes shorter. There are, however, secondary irregularities of great importance which must be remembered, and which ought to influence the selection of operation in any particular case. Some operations, if performed in an ideal manner, give good results; other operations, no matter how perfect the technique, or how gainly the stump, must be put down as failures. Then again the appliances required for some amputations last without repairs for years, whilst others are constantly needing attention. All these points are of material importance in the case of an employed workman, not to mention the fact that amputations as done are rarely perfect. They may show a badly placed, or adherent and tender, scar; they may contain a painful nerve bulb; or a stiff joint may handicap an otherwise good result. Some of these defects cannot be foreseen, and therefore cannot be considered in mapping out the curve of capacity for work in general. The variants which can be considered, and which decide the relative values of adjacent amputations, are: (*a*) The value of the ideal amputation at the given level; and (*b*) The type of prosthesis appropriate to each particular variety of amputation.

Following the graph (*Fig.* 315) from left to right, we find that comfortable progression, and hence capacity for doing work, are diminished by an amputation of the great toe. Loss of the other toes does not as a rule produce any disability, though it is advisable if possible to retain them.

With a Lisfranc there is a marked drop in the capacity curve. This is, as has been shown, primarily due to the shortening of the anterior portion of the arch and its consequent flattening. The secondary changes are, however, of even greater importance. These are due to the muscular action in trying to prevent deformity, and to the painful rigidity of the muscles involved. In many cases this amounts to spasmodic inversion of the foot.

There is a still further drop in the curve with Chopart's amputation. Trèves regards this as the worst of all amputations in front of the ankle-joint. The reasons for this have already been explained. There is the complete removal of the anterior pillar of the arch ; the bones are rotated round a transverse horizontal axis, so that their anterior aspects come to be the chief bearing surface ; the scar, at first supero-anterior, follows the bones downwards until it comes in contact with the ground ; some degree of varus usually complicates the potential equinus ; and added to these defects we have an artificial appliance which is constantly in need of repairs.

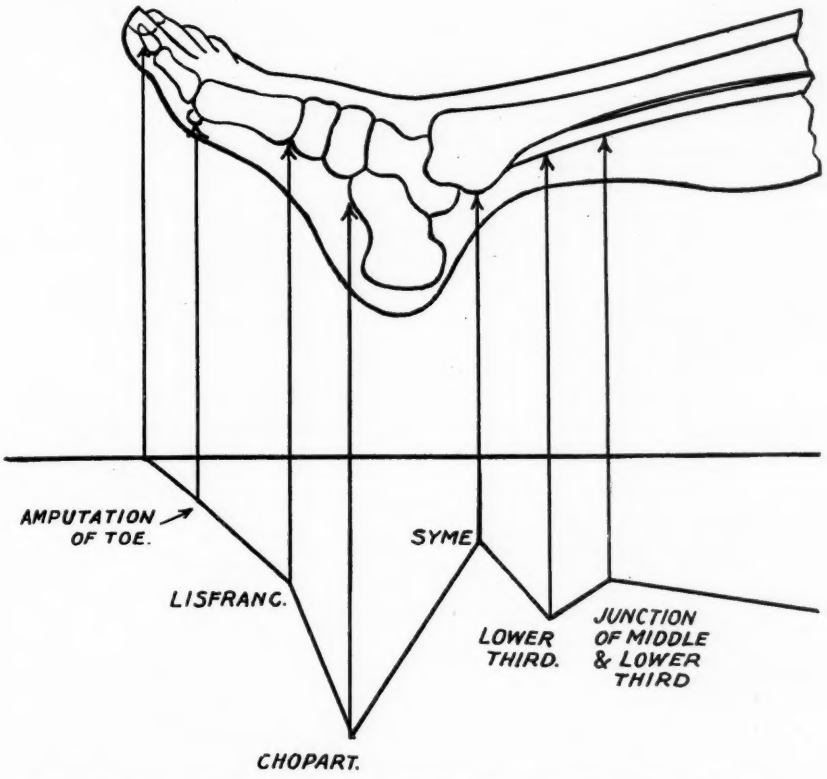

FIG. 315.—Graphic representation of capacity for work in partial amputations of the foot.

Between the classical operations of Lisfranc and Chopart the degree of comfort and the capacity for work vary. The further forward the amputation, the greater the value of the operation.

With Syme's amputation we find an abrupt rise in the capacity curve, indicating that the ideal performance of this operation leaves a stump which is, from the point of view of function, as nearly perfect as possible. It gives a complete and permanent end-bearing ; there is a long lever for the activation of the artificial limb and the provision of perfect stability ; and the presence of the natural knee gives almost, if not indeed quite, perfect gait. The only criticism to be offered is that the artificial ankle, owing to the side bars, is rather bulky and *disgracieuse* (Trèves).

CONCLUSION.

The conclusion come to as the result of this investigation of partial amputations of the foot is that, unless more of the limb can be saved than that found in Lisfranc's amputation, the function of the limb, as estimated by the man's capacity for work, his

ability to keep time, and his value as a unit in the general industrial machine, will be improved by the complete amputation of the foot through the ankle-joint after the manner of James Syme.

* * * * *

My thanks are due, and are hereby expressed, to Mr. P. T. Crymble and Dr. R. J. McConnell for the radiograms, to Dr. H. P. Hall for the photographs reproduced in *Figs.* 311 and 313, and to Mr. R. W. H. Blackwood for the drawing of the graph.

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THE HÆMANGIOMA GROUP OF ENDOTHELIOBLASTOMATA.

By JOHN FRASER, Edinburgh.

While working in the Out-Patient Department of the Children's Hospital, I have been impressed by the large number of children who are brought for treatment on account of hæmangiomata. In the majority of the cases the tumours appeared in relation to the skin and subcutaneous tissue. In a comparatively small proportion they have developed in relation to the mucous membrane, more especially of the mouth, tongue, and rectum. There has been a group of rare cases in which hæmangiomata occurred in the following situations : voluntary muscle, subsynovial tissues (knee-joint), liver. (*Figs.* 316–320.)

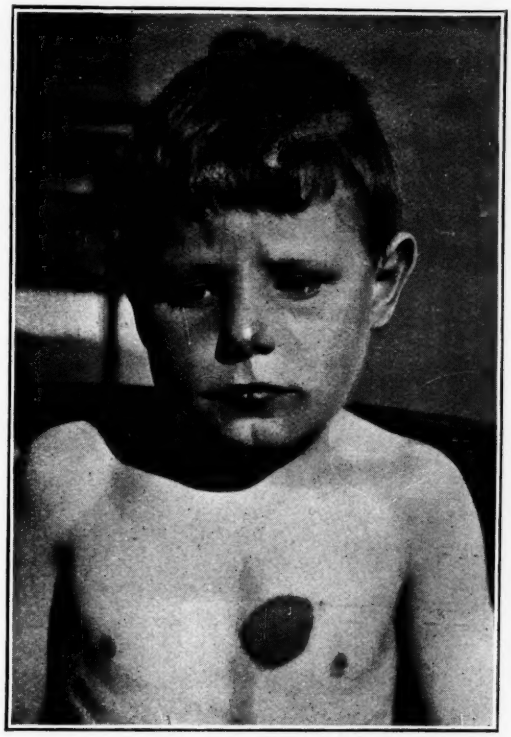

Fig. 316.—Cutaneous hæmangioma of the left breast.

Fig. 317.—Multiple hæmangiomata of the tongue.

Fig. 318.—Cavernous hæmangioma of the face.

The interest which these cases aroused led to a clinical and histological investigation of them. The literature dealing with the subject is scanty, and in many cases the description from a histological point of view is erroneous. It is apparently not sufficiently recognized that these are definite

tumours, with an ever-present tendency to infiltration of the surrounding tissues, and occasionally giving rise to metastasis.

There are many problems which arise in connection with these tumours: questions of their origin, their evolution, their classification, and their histological characteristics. With these problems I have attempted to deal.

ENDOTHELIOBLASTOMATA.

These may be defined as tumours of mesenchymal origin, the cells of which tend to differentiate into flat endothelial cells such as line the interior of blood- and lymph-vessels and the inner surface of certain cavities or spaces.

Under such a definition three different types of cells are recognized: (1) Endothelial cells lining blood-vessels; (2) Endothelial cells lining lymph-vessels; (3) Endothelial cells lining the arachnoid or subdural spaces.

The endothelial cells lining the heart, the blood-vessels, and the lymph-vessels have a common origin, and they may be considered identical in all respects; but the cells lining the arachnoid and subdural spaces are derived from the mesenchyma at a considerably later period, and, in certain other particulars with which we need not deal, they differ from

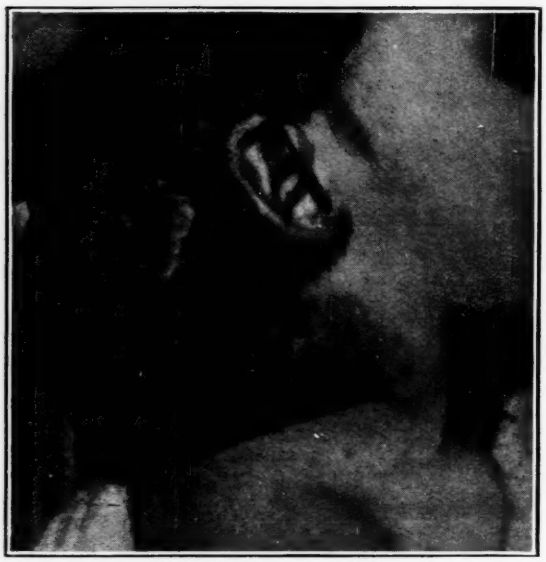

FIG. 319.—Large compact hæmangiomata of the neck.

FIG. 320.—An extensive hæmangioma of the left cheek.

the first-mentioned group. It is with the type of tumour derived from the endothelial cells lining blood-vessels that this communication is concerned—the so-called hæmangio-endothelioblastoma.

Terminology.—At present there is much confusion in the terminology which is applied to the condition. Frequently the term nævus is used. Now this word strictly means a mole or natural mark, and in the Continental literature, following Ribbert's teaching, the term is strictly applied to these pigmented tumours, which are classified under the melanomas. It would be well, therefore, and it would certainly avoid confusion, if the term hæmangioma were used to the exclusion of all others as the descriptive title of the condition which we are discussing: and further, the term should be limited to those tumours in which there is an actual new formation of vessels, or a proliferation of the vessel walls, aneurysms and varicose veins being thus excluded.

I am anxious to make it quite clear that I am not concerned with those tumours of the skin which contain the so-called nævus cells. They are essentially of the types of tumour which are properly called moles, and they have no relationship to the hæmangiomata. In a recent article in the *British Journal of Dermatology*, Fox[1] has entered fully into the histological characters of the so-called moles, and his conclusion

agrees with those originally advanced by Unna[2], that the nævus cells are epidermal in origin, being derived in all probability from the 'prickle cells' in the deeper layers of the skin. In the hæmangiomata the cells which constitute the characteristic tumour are derived from endothelial cells, and therefore have no connection whatsoever with the nævus cells of epidermal origin. The pity is that the term nævus should be so loosely applied as to be used to describe hæmangiomata ; and much confusion will be avoided if the term is limited to tumours containing true nævus cells.

The present system of subdivision of the hæmangiomata calls for comment. In many text-books the classification is adopted of capillary, venous, and arterial ; but if our definition of the term hæmangioma is to hold good, namely, a tumour in which there is an actual new formation of vessels or proliferation of the vessel walls, this classification is no longer tenable. These tumours originate from the endothelium lining the vessel wall, and, moreover, in every instance the endothelium is capillary in origin. It is more reasonable, therefore, that the classification of hæmangiomata should depend upon the variety of histological structure, and more especially upon the density of its structure. On such a basis the most suggestive classification would be one which divides these tumours into compact, capillary, and cavernous types. The reasons for the subdivision become more obvious when we discuss the various histological features. The classification which is based upon the relative positions of the tumour is quite reasonable from a descriptive point of view. It subdivides the tumours into cutaneous, subcutaneous, and mixed varieties.

FIG. 321.—Low-power appearance of capillary hæmangioma (*camera lucida drawing*).

Origin of the Tumours.—These tumours are congenital in origin. The first vessels which appear in the embryo are formed in the mesoderm ; and development goes on subsequently in the same layer and in its derivations in all parts of the animal body. The vessels are originally formed in the vascular area of the embryo. Certain cells of the mesoderm (angioblasts) become vacuolated, and proliferate in such a way as to form a syncytium. Fluid collects within the vacuoles, which, enlarging and proliferating, give rise to minute reddish specks, the so-called 'blood islands of Pander.' The enlarged parts of the syncytium are united to one another by narrower parts, and after a time the cavities extend into the narrow portions, so that a network is produced. The wall of these primary vessels is composed at first merely of the protoplasm of the syncytium, with nuclei embedded in it here and there. Subsequently, the protoplasm becomes differentiated around the nuclei into the flattened cells which compose the walls of the capillaries, and which form the lining walls of the arteries and veins.

Everything points to the fact that it is from 'rests' of this embryonic tissue that hæmangiomata are developed. It may be urged that this explanation does not account for the small multiple angiomata which appear after middle age, the so-called 'de Morgan's spots.' There is, however, nothing unlikely in the fact that they have a similar explanation ; though possibly they may originate from the endothelium of fully-developed capillaries as a result of degenerative changes in the capillary wall, and the escape of endothelial cells beyond the confines of the capillary lumen.

Histological Structure.—The endothelial cell is not morphologically a highly differentiated cell, and on this account it stands out in contrast with other more characteristic cells. Individually, it is a large flat cell with an oval nucleus surrounded by a moderate amount of delicate cytoplasm. Under normal conditions these cells possess marked phagocytic properties, and they may desquamate and give rise to endothelial leucocytes ; but in tumour formation they possess no such distinctive properties.

The Development of the Tumour.—From the original site of development the endothelial cells proliferate, and by their proliferation and arrangement they tend to form blood-vessels, as they do under normal conditions. The simplest form of hæmangioma therefore consists of a collection of embryonic blood-vessels. The endothelial cells which form the walls are of relatively considerable thickness, averaging 3 to 4 μ; and in this respect they differ markedly from the endothelial cells which line fully-developed capillaries. The cells are nucleated, and many of them show active division and numerous mitotic figures. The vessels are backed by a very slight amount of connective tissue, which forms a stroma and binds them together. Sometimes the stroma is abundant, so that the tumour appears more or less scirrhus-like in type. The lumen is generally empty; occasionally it contains a few corpuscular elements, but they appear to be either immature or degenerate.

FIG 322.—High-power appearances of a group of elements of a capillary hæmangioma (*camera lucida drawing*).

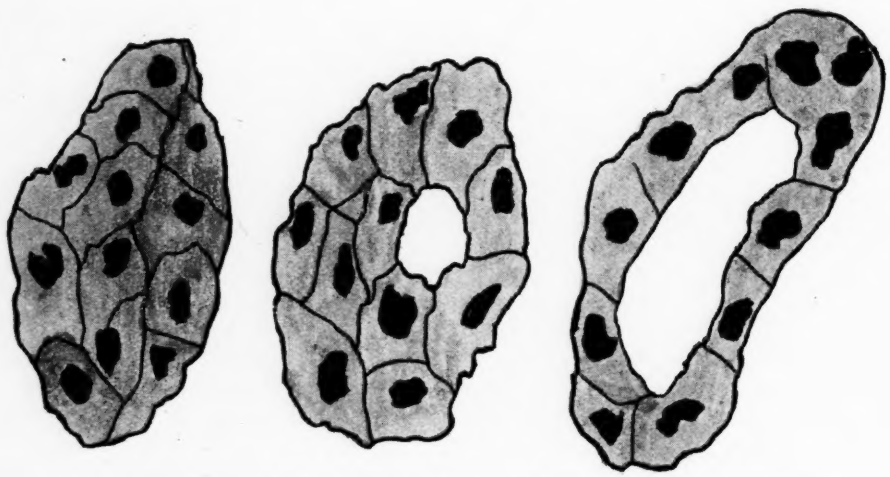

FIG. 323.—Successive stages in the formation of a capillary hæmangioma (*camera lucida drawing*).

Active proliferation of the endothelial cells is always occurring, and it is possible to demonstrate the development of fresh formation of vessels. Within the accumulation of several endothelial cells which have proliferated from the walls of an existing vessel, a vacuole makes its appearance, and by its enlargement establishes the lumen of the future vessel. Around the central vacuole the endothelial cells become so arranged as to form the vessel wall. The repetition of this process leads to the development of further collections of embryonic vessels (*Figs.* 321–324).

FIG. 324.—The appearance with a high power of a single capillary element of the tumour (*camera lucida drawing*).

The further extension of the tumour is upon an infiltrative scheme. If the tumour is developing in the subcutaneous tissue, it surrounds the coil glands and ducts, and it invades lobules of fat tissue, nerves, and muscle fibres : nerves especially are very often enlarged and infiltrated.

There is little that is peculiar about the blood-supply. The tumour tissue is well supplied with blood : the smaller arteries, veins, and capillaries circulate between the tumour elements ; but it is important to note that the lumina of the cells do not communicate with the general blood-stream, except under special conditions which we shall mention presently. The supplying vessels run in the connective-tissue stroma supporting the tumour cells.

The Further Evolution of the Tumour.—There are at least four possible and different directions in which the evolution of the tumour may occur : (1) Its growth may become arrested, the tumour eventually undergoing spontaneous cure by a process of fibrosis ; (2) The tumour, while retaining the characteristics of a capillary hæmangioma, continues to grow by a process of infiltration of the surrounding parts ; (3) The original capillary type of hæmangioma becomes converted into a cavernous type of hæmangioma ; (4) The original capillary type of tumour becomes converted into what we have termed the compact type of hæmangioma.

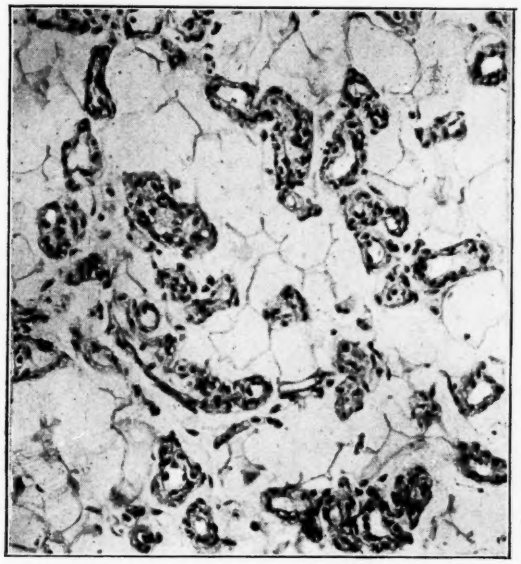

FIG. 325.—Appearance of infiltrating capillary type of hæmangioma (×160).

FIG. 326.—Edge of subcutaneous capillary hæmangioma (× 60).

1. **Natural Arrest and Spontaneous Cure.**—It is fortunate that in these tumours there is a strong and insistent tendency towards natural cure. It would appear that the process of cure depends upon a deficient and a diminishing blood-supply. The connective-tissue stroma—which, in greater or lesser quantity, always surrounds the tumour elements—is converted into a dense fibrous tissue. By a perivascular and endovascular thickening, the blood-vessels undergo a progressive obliteration. The diminished blood-supply, and the pressure of the surrounding fibrous tissue, eventually lead to a complete disappearance of the tumour tissue, its position being replaced by fibrous tissue. The various therapeutic measures which have from time to time been suggested and employed have as their object the hastening of this natural process of cure.

2. **Progressive Spread and Infiltration.**—The extensive infiltration of which this tumour is capable is probably insufficiently recognized. Certain clinical cases have been recorded which can only be accounted for by the property of infiltration. Bormann[3]

reports the case of a hæmangioma occurring in the breast which repeatedly recurred after excision, and which finally produced metastasis in the lungs. Mallory[4] describes a case which started as a hæmangioma of the eyelid, and which progressively invaded the orbit and eventually destroyed the eyeball.

The distribution of the infiltration can be best studied in a hæmangioma occurring in relation to the skin (*Fig.* 325). The most marked infiltration occurs in the cutis vera : the most superficial layers of the skin are rarely invaded (*Fig.* 326). The coil glands and ducts, while they are separated and surrounded by the tumour, are not individually invaded (*Fig.* 327).

Infiltration is extensive in the subcutaneous fatty tissue, the tumour extending between individual fat cells, and among the fibres of the connective-tissue stroma (*Figs.* 328 and 329). The deeper the examination is carried, the less marked does the infiltration become. To some extent infiltration occurs in muscle, the tumour extending between individual muscle fibres. We have already remarked upon the degree to which nerve

FIG. 327.—Infiltration of capillary hæmangioma around coil glands of skin (× 60).

FIG. 328.—Infiltration of subcutaneous fat by capillary hæmangioma (×160).

tissue is involved : the sheath of the nerve is invaded, and there is a spread inwards between individual nerve fibres.

It is interesting to observe the exact mode by which the infiltration occurs. The tumour spreads, not by the embryonic capillary tissue which one finds in the fully-developed growth, but by individual endothelial cells. These afterwards proliferate and become vacuolated, eventually forming the embryonic capillary tissue.

3. **The Development of the Cavernous Type of Hæmangioma.**—If the embryonic capillary tissue develops a connection with the circulation, the cavernous type of hæmangioma may result (*Fig.* 330). The original embryonic capillary vessels become distended, probably from the passage through them of the circulating blood under some degree of pressure. The lining endothelium becomes very much thinner, and the cavity is filled with blood, which, judging from the character of its corpuscular elements, is in active circulation. In this last respect the contents of the cavernous hæmangioma differs very markedly from that of the capillary type, the corpuscular contents of which are either imperfectly developed or degenerated. It is the exception to find a tumour in which the cavernous change has become general : in almost every instance, if the cavernous

tumour is present, it is associated with the capillary type, and with varying stages of transition between the two.

4. **The Development of the Compact Type of Hæmangioma.**—If, for some reason, the endothelial cells lining the capillary type of tumour take on active proliferation,

FIG. 329.—Capillary hæmangioma showing extensive infiltration of related subcutaneous fat (× 60).

FIG. 330.—Cavernous type of hæmangioma (×150).

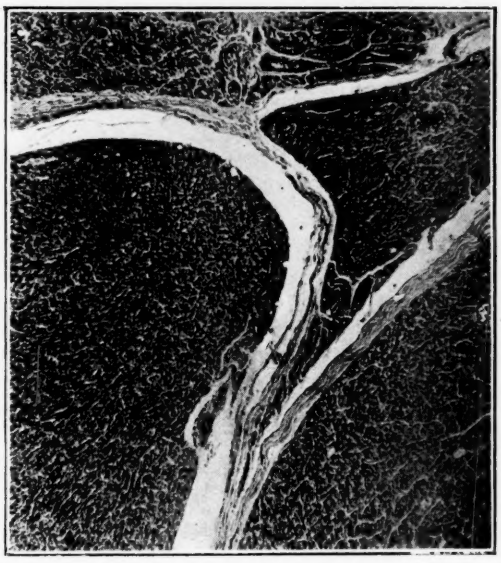

FIG. 331.—Appearance of compact type of hæmangioma (×50).

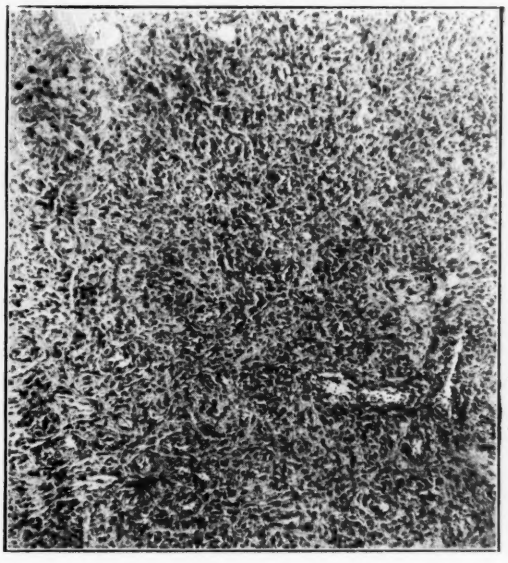

FIG. 332.—High-power photograph of compact type of hæmangioma (×200).

the compact variety of hæmangioma may develop (*Figs.* 331 and 332). No explanation can be offered to account for the onset of the proliferation. The multiplying cells do not form vessels, but instead collect in rows, masses, whorls, and groups. Generally the proliferation is perivascular in type; occasionally it is endovascular, the cells projecting

in papilla-like arrangement, and becoming arranged in concentric masses and whorls. The development of the compact variety of hæmangioma is accompanied by a localization of the tumour: it is very exceptional to find infiltration occurring in this type. There is a definite capsule of fibrous tissue encircling the mass, and from the outer envelope septa extend inwards, breaking up the tumour into a lobular arrangement.

The compact hæmangioma is well supplied with fully developed blood-vessels, which are carried in the fibrous tissue of the capsule, and extend inwards among the tumour-cells. The endothelial cells of the compact hæmangioma sometimes accumulate fat, and sometimes blood pigment. In this latter condition the pigment owes its origin to degenerated blood-corpuscles, which have been retained between the endothelial cells: the pigment, if large in amount, may give the tumour a yellowish appearance, and occasionally this leads to the tumour being mistaken for a xanthoma.

CONCLUSIONS.

1. Hæmangiomata constitute the most common variety of all the tumours which are met with during the period of childhood.

2. The tumours are congenital in origin, arising in all probability from 'rests' of embryonic vascular tissue.

3. The further development of the tumour leads to the formation of capillary, cavernous, and compact varieties.

4. The tumour has striking powers of infiltration, and occasionally it may give rise to metastasis.

* * * * *

I wish to acknowledge the assistance, by suggestion and criticism, which I have had from Dr. J. W. Dawson in the preparation of this paper.

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[2] Unna, P. G. *Histopathology*, p. 1129.
[3] Bormann. *Deut. Zeitschr. f. Chir.* 1910, p. 323.
[4] Mallory. *Pathologic Histology*, p. 309.

THE MORBID CONSEQUENCES OF A MOBILE ASCENDING COLON, WITH A RECORD OF 180 OPERATIONS.

By GEO. E. WAUGH, London.

The ascending colon is a segment of gut that is usually regarded merely as a segment of the large intestine, subject to the same disorders as the rest of the colon, and performing very similar functions. A closer study of its function, and of its anatomical relationships, reveals them both to be unique, and possibly of great surgical significance.

The uniqueness of its function, in association with its intimate anatomical relations, appears to provide the explanation of the existence of many abdominal diseases; of the course they run, from the early stages of comparatively trivial symptoms to the late and inevitable stage of gross morbid changes; and finally of their grouping in the right upper quadrant of the abdominal cavity. In this quadrant are situated the pyloric region of the stomach, the duodenum, the biliary apparatus, and the right kidney.

Abdominal Diseases.—A list of surgical diseases of the abdominal cavity drawn up without reference to the contents of this quadrant would include little save those of the appendix and rectum, and abdominal operations would so shrink in number as to lose their entirely preponderating position amongst present-day surgical procedures.

Surgical endeavour has been concentrated, for the last twenty years, upon the task of a successful modification of method and technique in the treatment of such end-stages of disease as gastric and duodenal ulcers, gall-stones, and prolapsed right kidneys. While meeting with an uncertain measure of success—an uncertainty that is revealed by the life history of gastrojejunostomy from its inception to the present time; by the rival claims of gastrojejunostomy against gastrectomy; by a preference for the use of the cautery instead of the knife for excision of a gastric ulcer, with renascent hopefulness about the issue; and by the hesitation that is experienced in attempting to decide between the merits of cholecystectomy and cholecystostomy—it has been subconsciously realized that the problem of the origin of these diseases has not been solved, that the treatment evolved has not been based upon that knowledge, and that an erratic distribution of failures and successes is an inevitable consequence of empiricism.

Nor have scientific investigations upon animals, in spite of patient and brilliant research, been able to throw much light upon the human problem, or to add much, from the knowledge thus gained, to the successful treatment of these diseases in human beings. By means of cytotoxins and attenuated bacteria, some of the diseases, in some measure, have been reproduced in animals, but so lacking in faithful mimicry of the human lesion that it is impossible to escape from the conclusion that some factor has been wanting from the experiments that has robbed them of complete success. This factor is possibly a mechanical one—one that would be difficult to improvise in the course of an experiment, but which is ready to hand in the human being, who is perpetually exposed also to the influences of bacteria and toxins. This factor, I venture to think, is resident in an abnormal mobility of the ascending colon; and by an appreciation of its significance some solution may be found of the general surgical problems which form the background for the study of these diseases. Some of these problems may be indicated briefly in the form of questions:—

1. Why are all these diseases grouped together in one small area of the abdominal cavity?

2. Why are they almost unknown in childhood?

3. Why does their onset occur so frequently about the age of twenty years and onwards?

4. Why are gastric ulcers clustered around the pyloric region of the stomach, and why is it that duodenal ulcers are almost always in the first and second parts of the duodenum ?

5. What are the prodromal stages of gall-stones, gastric ulcers, and duodenal ulcers ?

6. Why is the right kidney so frequently mobile in these diseases, and why is it the only mobile one in 80 per cent of all cases of movable kidney ? (Thomson Walker.)

7. Why does the right kidney so frequently prolapse again after very firm surgical anchorage ?

Mechanical Factor.—It is certain that, apart from some specific infective diseases, ulcers in most parts of the body—more especially the conjunctiva, tongue, septum of nose, legs, sacral and gluteal regions—are formed directly under the influence of a recognizable mechanical factor, and that healing can be brought about only when the mechanical irritant has been removed. The mechanical factor that leads to focal ulceration of the stomach and duodenum, to the stagnation of bile, and to mobility of the right kidney, may conceivably be provided by a mobile ascending colon ; and a consideration of its intimate anatomical relationship to these areas of disease, together with the uniqueness of its function, will reveal how this may occur.

Anatomical Relations of the Ascending Colon.—By the ascending colon in this article is meant that segment of the large intestine which is included between the lowest point of the cæcum and the highest point of the hepatic flexure (the identification of the cæcum apart from the colon has arisen with reference to its developmental origin and for purposes of descriptive anatomy ; from the standpoint of surgery and pathology, however, this separation is artificial, and is not followed in this article). This segment of gut lies deeply in the posterior part of the abdominal cavity, and ascends from the right iliac fossa, lying upon the iliacus and quadratus lumborum muscles to the anterior aspect of the right kidney, which it overlies, and thence bends inwards, at the hepatic flexure, across the junction of the second and third portions of the duodenum. The flexure lies immediately below the pyloric end of the stomach, and in intimate, but very variable, relationship with the gall-bladder and cystic duct. With the exception of the cæcum, it is plastered back securely into its position by the reflection of the peritoneum from its lateral aspects to the posterior abdominal wall, so that the posterior wall of the gut is bare of peritoneum and actually lies upon the structures already enumerated behind it. (*Fig.* 333.) From the hepatic flexure the peritoneum passes upwards over the duodenum and head of the pancreas to the lower border of the pyloric region of the stomach, and in many instances to the under aspect of the gall-bladder and cystic duct. (Detailed references to this last relationship are made further on.)

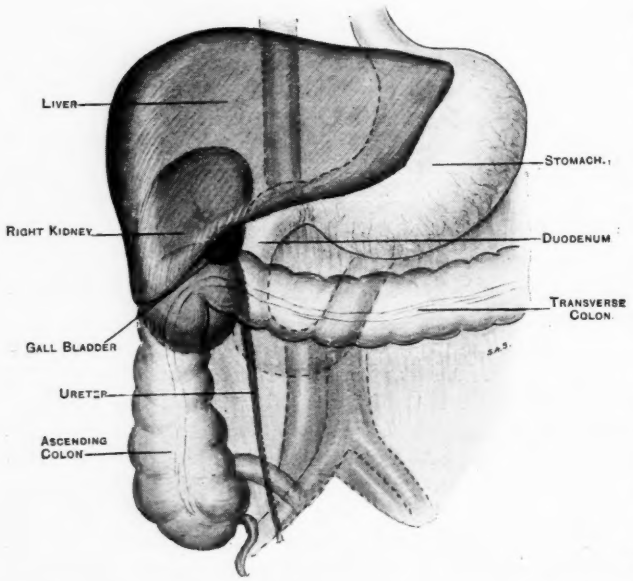


FIG. 333.—Diagram to illustrate the chief relations of the ascending colon and hepatic flexure.

Fixation of Ascending Colon.—This firm fixation of the ascending colon is a prime mechanical necessity for the efficient performance of its functions ; and in this connection it is important to note how this fixation has been attained. In the early stages of

development the ascending colon lies upon the left side of the body cavity in direct continuation with the primitive alimentary tract, and suspended by the same mesentery which is attached to the whole of the dorsal aspect of that tube. A rotation then occurs

FIGS. 334 and 335 (modified after Cunningham) illustrate the mode of development of the intestine and mesenteries. *Fig.* 334 is a lateral view showing the rotation of the large intestine from left to right around the axis of the superior mesenteric vessels. The cæcum is still high up in the region of the liver, and the primitive mesentery is still attached to the whole of the large intestine: (1) duodenum, (2) cæcum, (3) mesentery of small intestine, (4) rectum, (5) stomach, (6) primitive mesentery of large intestine. *Fig.* 335 is a front view after rotation is completed and the cæcum has nearly finished its descent to the right iliac fossa: (1) cæcum, (2) mesentery of small intestine, (3) transverse mesocolon, (4) sigmoid mesentery. The dark shaded areas (*a*) show where disappearance of the primitive mesentery of the large intestine normally occurs.

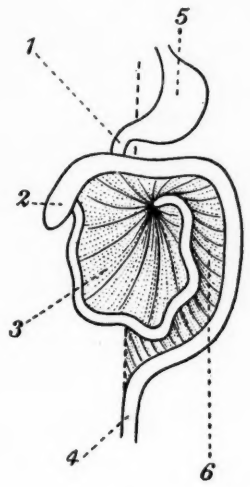


FIG. 334.

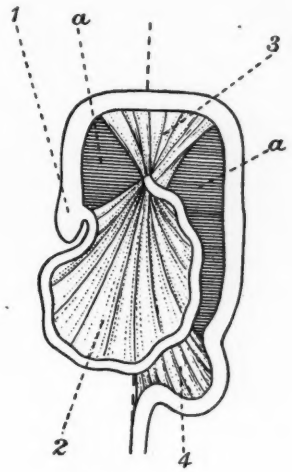


FIG. 335.

around the axis of the superior mesenteric vessels, whereby the part of the gut that is to become the ascending colon is swung across to the right side immediately below the liver, and eventually descends to that position which it normally occupies (*Fig.* 334). As soon

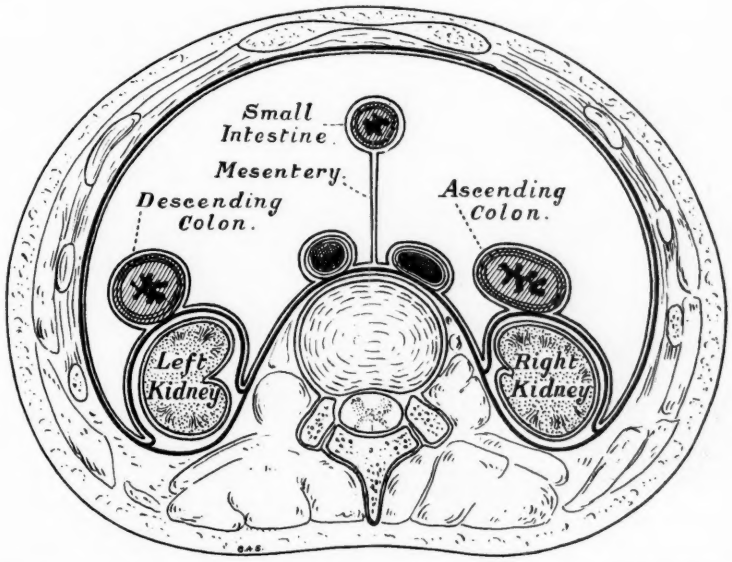


FIG. 336.—Transverse section across the lumbar region of the body to show the normal arrangement of the mesenteries.

as this is completed, its primitive dorsal mesentery disappears by fusion with the posterior parietal peritoneum (*Fig.* 335), and the fixation of the gut is completed, so that it is now held in position in the manner already described.

This is the normal course of development, but it is quite certain, from the study of

a large number of cases, that this does not always happen, and that the disappearance of the primitive mesentery is of most erratic incidence—an erraticism that is fraught with the consequence that the ascending colon is now a pendulous tube instead of a fixed segment of gut (*Figs.* 336 and 337).

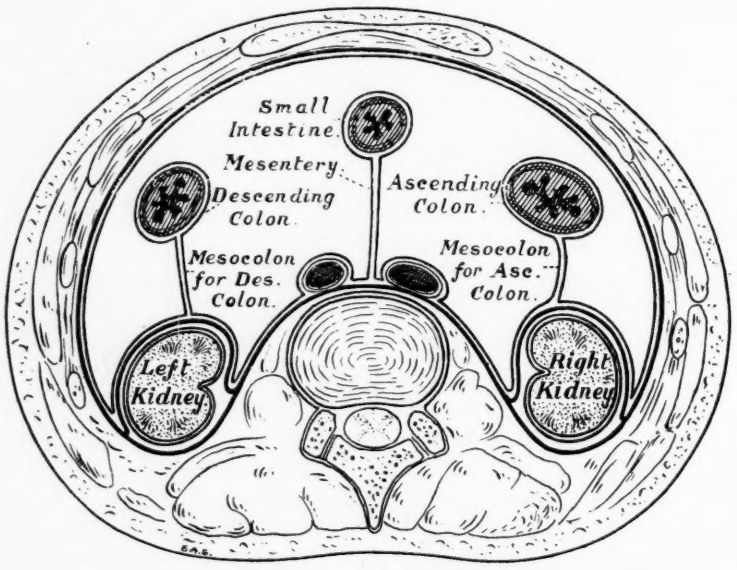


FIG. 337.—Transverse section across the lumbar region of the body to show the appearance when primitive mesenteries persist to the ascending and descending colon.

Abnormalities.—In the following series of cases upon which this study has been based, all types of irregularities in disappearance of the primitive mesentery have been noted—from the extreme type, at one end of the scale, where the whole of the large intestine lay

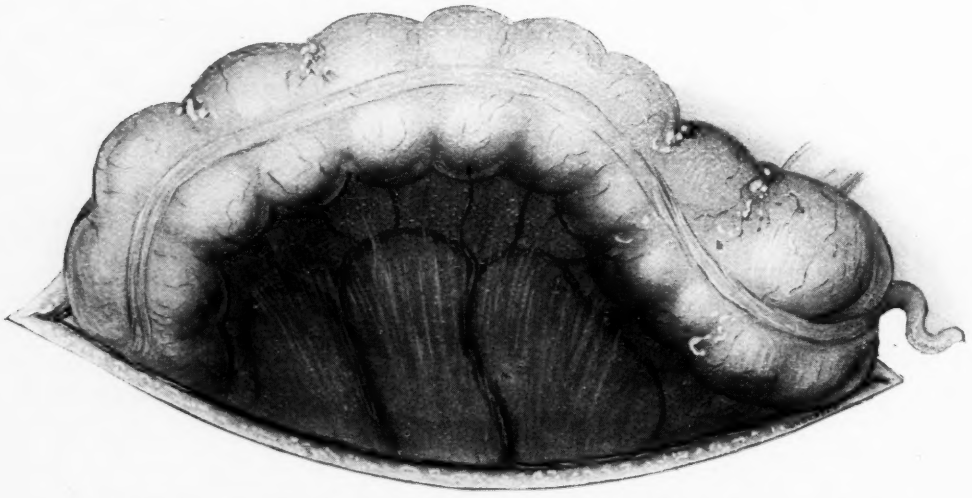


FIG. 338.—The ascending colon, from hepatic flexure to cæcum, lifted out of the abdomen on to the towels, revealing the whole of its primitive mesentery with arches of blood-vessels persisting.

in the left iliac fossa, with a complete primitive mesentery and with apparently no attempt at rotation towards the right having occurred, to cases at the other end of the scale in which all but a small segment of the primitive mesentery had disappeared, and with the

colon in its proper position on the right side of the body. The intermediate types, which comprise more than 95 per cent of the cases in this series, include such frequent variations as the following :—

1. With a right-sided ascending colon, a complete primitive mesentery persists from hepatic flexure to cæcum, bearing between its folds the vascular anastomosis between the ileocolic and the right colic arteries. The ascending colon in this group may be lifted out of the abdominal cavity entirely, and placed upon the towels covering the *left* side of the abdomen. This condition was found in 77 per cent of all cases in this series (*Fig.* 338).

2. The upper half only, or the lower half only, of the primitive mesentery may persist, with its respective blood-vessels lying between its layers.

3. Only a small segment of true mesentery is present : but a pseudo-mesentery dragged out from the fascia of the posterior abdominal wall is present in addition. This pseudo-mesentery is readily distinguishable from the true mesentery, with its characteristic vascular anastomosis seen through a fatty membrane, by its opaque white surface devoid of blood-vessels, and its linear anterior edge directed towards the colon and continuous with the posterior edge of the true mesentery.

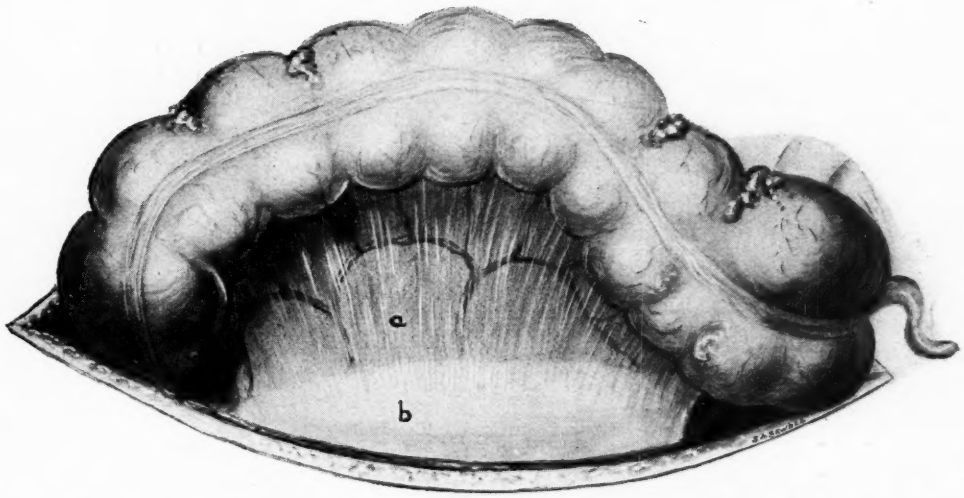


FIG. 339.—The whole of the true mesentery (*a*) is present, and a pseudo-mesentery of fascia (*b*) has been dragged out along its posterior line of attachment.

4. The whole of the true primitive mesentery is present, but along the whole length of its posterior attachment a pseudo-mesentery of fascia has been dragged out (*Fig.* 339).

5. The lower half only of the true mesentery is present, while the upper half of the colon is suspended on a pseudo-mesentery of fascia, and vice versa (*Fig.* 340).

6. Portions of the upper and lower half of the true mesentery are present, with the intervening segment of gut swung upon a pseudo-mesentery of fascia.

7. Rotation of the gut from left to right has occurred, but the complete descent of the cæcum has not been accomplished. In these cases the ascending colon is represented by a comparatively short segment of gut swung upon its primitive mesentery, with the lowest border of the cæcum barely reaching to the level of the iliac crest.

8. In two cases, with a completely mobile ascending colon, the whole of the small intestine in addition was entirely retroperitoneal, and was not visible except through this transparent membrane throughout the course of the operation of fixing the colon. The terminal part of the ileum made its appearance over the left brim of the pelvis through a foramen in this sheet of peritoneum, to the margins of which it was firmly attached. This sheet of peritoneum is dragged across the abdominal cavity by the large intestine during

its rotation from left to right, and should then disappear. Its failure to do so results in the above condition with the small intestine contained in a closed sac of peritoneum, whose margins are firmly fixed to the wall of the large intestine.

These variations in the position of the ascending colon, and in the persistence of its primitive mesentery, appear to represent arrested stages of development, such as might be expected from the mode of development of the colon and its mesenteries, whilst the appearance of the pseudo-mesentery of fascia is an acquired condition which is the direct consequence of the persistence of the primitive mesentery. That this is so, is apparent from the comparative study of the ascending colon in children and adults.

In children under twelve years of age a pseudo-mesentery of fascia is hardly ever found, while from a series of post-mortem examinations of several hundred cases kindly conducted for me by Dr. Pirie at the Hospital for Sick Children, Great Ormond Street, it was elicited that a primitive mesentery or a portion of it was present in 20 per cent of all cases examined, irrespective of the diseases from which they died. The converse, that 80 per cent started life with no remains of a primitive mesentery, i.e., with a fixed ascending colon, is surely not devoid of significance. At this stage too it may be noted as a point of great importance that general visceroptosis in children is extremely rare—three proven

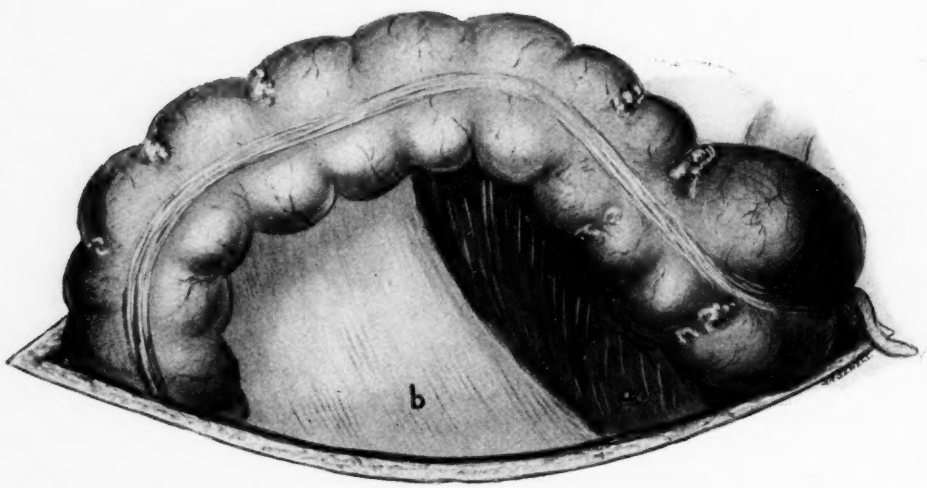


Fig. 340.—True mesentery (*a*) attached to the lower part, with pseudo-mesentery of fascia (*b*) attached to the upper part, of the ascending colon.

cases have come under the notice of the writer during the last fifteen years—and that it must be regarded as an acquired condition, making its appearance in adult life as the result of some slowly-acting force. This, too, may be correlated with the observation that the right kidney is rarely mobile in children, while it was mobile in 79 per cent of all the cases in this series of adults.

9. Variations in the omentum were also frequently noted. Its attachment to the transverse colon was continued over the hepatic flexure and down the anterior surface of the ascending colon to the extent of its upper half. This appears to be either an excessive development of the omentum, or an excessive descent of the transverse colon; but the resultant is a fold of omentum that passes as a tie-band between the upper half of the ascending colon and the right portion of the transverse colon, causing an abrupt angulation at the hepatic flexure, with increased obstruction to the outlet of the ascending colon.

Function of the Ascending Colon.—It has already been stated that the function of the ascending colon is unique. *It is the only segment of gut throughout the alimentary tract that has to support semi-solid material against the action of gravity and to drive it vertically up-hill.* Elsewhere the contents are fluid, or the path to be followed by them is

either obliquely horizontal or down-hill. Mechanical efficiency for this task can only be obtained with a fixed segment of gut such as the ascending colon should be after the complete disappearance of its primitive mesentery. The weight both of the gut itself and its load is then distributed sideways through its broad lateral attachments, and its normal function can be carried out without any shifting of position.

But with the persistence of its mesentery, or a part of it, the gut must sag until the mesentery is taut, and as the gut receives its normal load, so will the tautness of the mesentery increase. Further, the wave of peristalsis starting in its lowest segment will expend its energy partially on the mobile segment of the gut immediately above it, and partially upon its contents, instead of entirely upon its contents as it would if the gut were fixed. The whole strain exerted by the weight of the gut and its contents is now concentrated along the narrow linear attachment of its primitive mesentery posteriorly, and the tendency to progressive overloading is established through the wasteful expenditure of the wave of peristalsis in moving the gut as well as the load.

Such a mobile ascending colon is mechanically inefficient for carrying out its normal function ; and the simple conception of disease as a primary structural deficiency leading to functional disability is achieved. There is no need to hypothecate a primary and apparently spontaneous functional disability making its appearance and leading eventually to structural alteration ; the structural deficiency is here from the first ; it is severely conditioned by the laws of gravity ; and its origin is clearly comprehended by reference to the mode of development of the large intestine. Just as the heart cannot perfectly adjust itself to the physical laws conditioning the circulation of the blood if the foramen ovale is not closed at birth, so the ascending colon cannot perfectly adjust itself to the strain made upon it by the laws of gravity in performing its normal function so long as it is a mobile and not a fixed viscus.

A complete maximal inefficiency, however, will not be established from the beginning ; that will only be experienced when the gut, in passing from childhood to adolescence, has achieved its largest size and contains its heaviest load. No proportionate strengthening of the primitive mesentery occurs during this period, and the mesentery of adult life would be just as incapable of maintaining the ascending colon of a child in its proper position as the mesentery of that earlier period. An increase in the deposit of fat between its layers is perhaps the most noticeable change ; an addition that clearly is quite incapable of materially increasing the mechanical efficiency of supporting a segment of gut. Whilst, therefore, the disabilities resulting from this mobile condition in childhood, with its small gut and small load, are likely to remain obscure and masked unless especially sought for, they are likely to become gross and obvious as adult life is approached, from the fact that, while the supporting mechanism has gained nothing in strength, the load to be supported has enormously increased.

This appears, then, to be the explanation of the absence from children of the diseases already referred to ; of their tendency to appear from the age of twenty years and onwards ; of the characteristically progressive course of their symptoms, to be described in detail later ; and of the striking fact, in the histories of nearly all the cases in this series, that no detectable functional disability preceded any of their troubles. They were not peculiarly the victims of bad feeding ; there was no life-long history of indigestion or constipation ; they had not been especially exposed to bad environment or over-work ; they had not suffered from a devastating series of other illnesses ; in fact, no undue calls had been made upon the functions of the alimentary tract. The alimentary tract in these cases is revealed as obviously incapable of meeting very ordinary and seemingly normal demands upon its functional efficiency.

A very large number of the patients gave a history of a perfectly healthy childhood and early adolescence, and it had come as a great blow to them that with all the advantages they enjoyed they were not to remain the possessors of 'perfect health.' On the other hand, a few had suffered from 'digestive disorders' in childhood sufficiently vague to have prevented any greater precision in diagnosis than is contained in that phrase, and sufficiently baffling to have precluded any benefit from the varieties of treat-

ment heaped upon them. By the time that adolescence was reached, pain had generally become an added feature to their 'digestive disorders,' general malnutrition was evident throughout their tissues, and they were henceforth regarded as 'delicate people.'

The symptoms to which the growing mechanical inefficiency of the mobile colon may give rise in children will be discussed later under their proper heading. The local effects upon the viscera with which the ascending colon is in anatomical relationship must now be considered.

Local Effects.—The full weight of the gut with its load is distributed along the narrow line of the posterior attachment of its mesentery, instead of being dispersed over a broad area sideways, as when no mesentery is present. This strain falls directly upon the anterior surface of the right kidney, the second part of the duodenum, and, by radiation, upon the pyloric region of the stomach, the cystic duct, and, in some cases, upon the under aspect of the gall-bladder (*vide* gall-bladder cases and persistence of special band, p. 361).

It may be claimed that this strain can exert no injurious effect—an objection that can only be refuted by showing that, through the removal of the strain, the symptoms alleged to arise from it disappear. This series of cases seems to provide the necessary

FIG. 341.—Shows the second part of the duodenum sufficiently mobile to be grasped between the fingers and brought out of the abdominal cavity to the level of the skin incision.

refutation. But further, viewed merely as a problem in mechanics, two conditions may conceivably arise along the line of strain. Either the viscera pulled upon may be fixed in position so securely as to exert an equal and opposite force to the strain, when its full effect will be expended upon their tissues, leading to their destruction at the point of maximal strain, or, with a fixation less secure, they will advance along the line of strain and be pulled out of their normal positions, at the same time protecting their tissues from the full force of the strain.

The former condition is illustrated by the presence of gastric and duodenal ulcers in association with a mobile ascending colon; whilst it is merely a matter of observation at operation to determine that the latter condition is an extremely common one—even before operation it has been found that the right kidney was mobile in 79 per cent of all these cases, whilst x rays with barium meals as frequently showed the descent of the stomach; at operation, the mobility of the second part of the duodenum can be demonstrated with ease, since it can be picked up between the fingers, and brought up to the level of the incision in the skin (*Fig.* 341). This has been possible in nearly all cases of 'hunger pain' included in this series, while it has never been possible in the few cases in

which a duodenal ulcer was actually present. In the few cases with a fixed duodenum and 'hunger pains' but no ulcer, presumably the operation has been done in a stage prodromal to ulcer formation.

The descent of the right kidney and of the pyloric region of the stomach are of course obvious at once. The hepatic flexure of the colon, together with the rest of the ascending colon, can be easily lifted out on to the towels, and gentle tugs upon it will cause the peritoneum passing upwards from its highest margin to pucker into folds, radiating the lines of strain to the duodenum (*Fig.* 342), gall-bladder and cystic duct, and the pyloric region of the stomach, including the free edge of the gastrohepatic omentum.

The mobility and capacity for prolapse of the hepatic flexure of the colon will have an important influence upon the position and direction of the transverse colon, and consequently upon the path of its contents. The normal levels of the hepatic and the splenic flexures of the colon are stated by Cunningham to be the tip of the tenth right costal cartilage and the space between the seventh and eighth left costal cartilages respectively.

FIG. 342.—The main lines of strain upon the duodenum are shown by the puckered folds of peritoneum which are raised up when the hepatic flexure is drawn gently downwards.

Between these two fixed points the transverse colon passes, slung by its own mesocolon to the posterior aspect of the abdominal cavity, and by the great omentum to the greater curvature of the stomach (*Fig.* 343).

As the hepatic flexure slides down to below the level of the iliac crest, the direction of the transverse colon will be altered in varying degree from the obliquely horizontal across the abdomen, which is its normal direction, to a sloping vertical on the right side, culminating in a long steep vertical ascent to the splenic flexure on the left side (*Fig.* 344); so that now a second vertical drive for semi-solid material has been incorporated in the path of the alimentary tract, with its attendant disadvantages and with the strain of its load falling upon the cardiac end of the stomach.

The splenic flexure but seldom prolapses out of its position, on account of its firm anchorage by means of the costocolic ligament, so that the cardiac end of the stomach is now exposed to the influences that demand either a prolapse from position, or focal tissue destruction where the strain is met by a resistance of equal intensity. In my experience, ulcers in any part of the stomach are comparatively rare in proportion to the number of

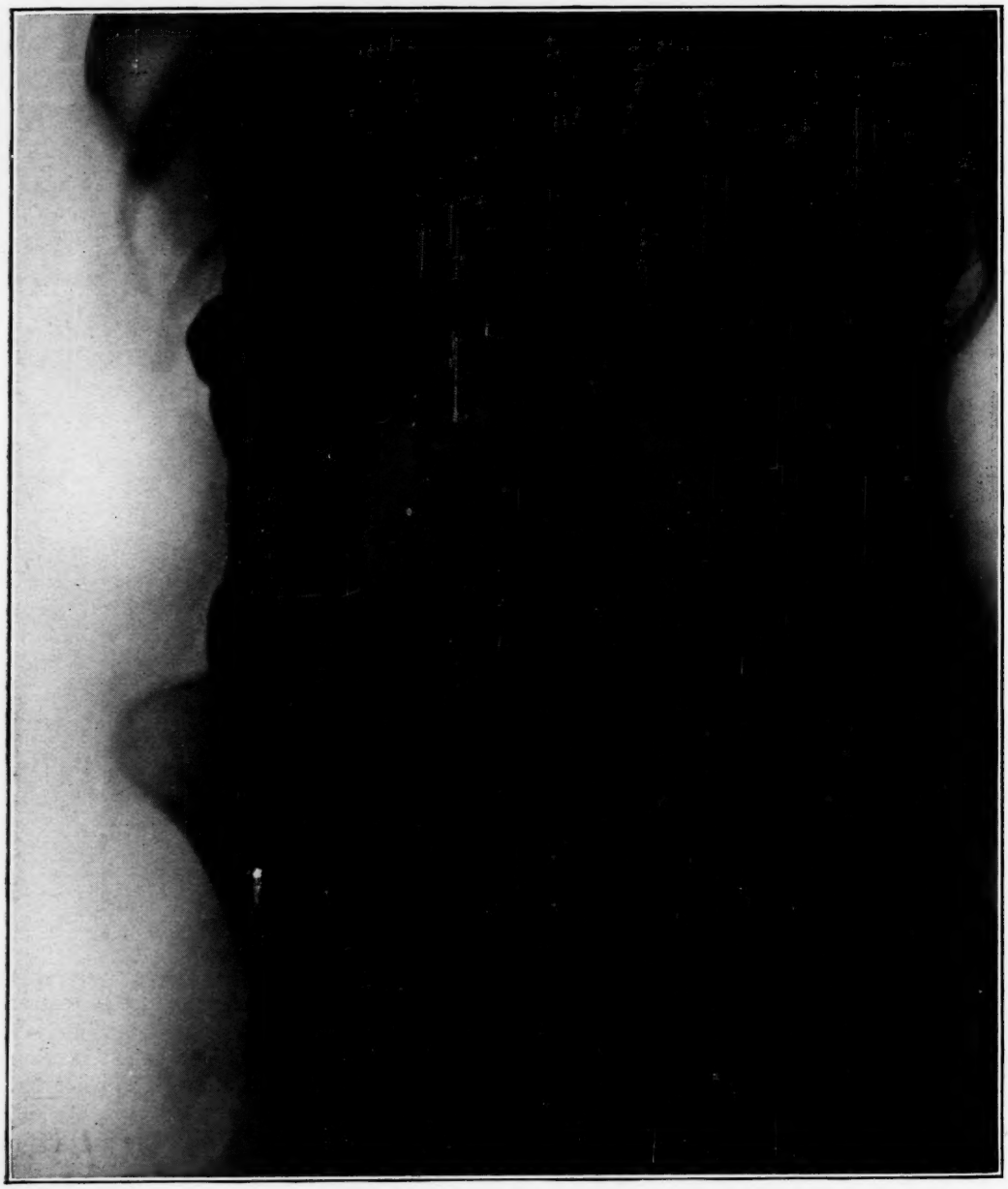


FIG. 343.—Radiogram from apparently healthy adult, aged 27 years, showing the normal position of the hepatic flexure and ascending colon, with the horizontal path of the transverse colon.

cases with true gastric pain, and this appears to be correlated with the ease with which prolapse of the stomach can occur. A low position of the stomach is one of the commonest features revealed by *x* rays and confirmed by subsequent operation (*Figs.* 345 and 346).

FIG. 344 (*Case* 107).—*Gastric type*, taken four hours after barium meal in the erect position at the end of inspiration. The hepatic flexure lies here just below the level of the iliac crest, whilst the steep vertical ascent from the transverse colon on the right side to the fixed splenic flexure is clearly defined.

FIG. 345.—*Gastric type*, eight hours after meal; erect position at end of deep inspiration; meal still partially in stomach. True mesentery to lower half of ascending colon, pseudo-mesentery to upper half. No organic disease of stomach detected at operation.

FIG. 346.—Shows characteristic low position of the stomach taken in the upright position at the end of deep inspiration ten minutes after meal. Patient had constant pain in right iliac fossa, with a severe dragging pain in right hypochondrium.

FIG. 344.

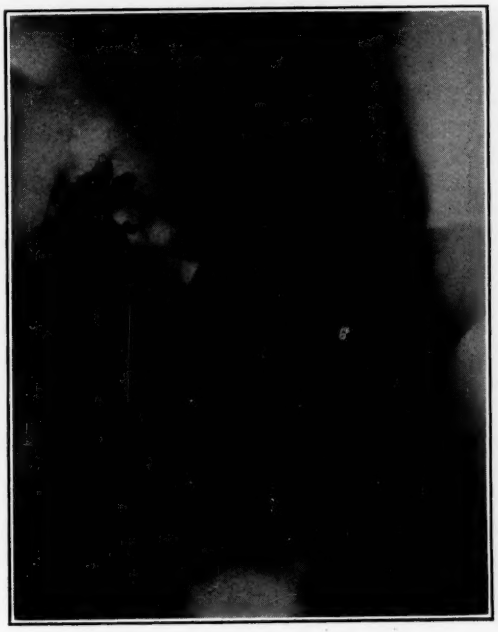


FIG. 345.

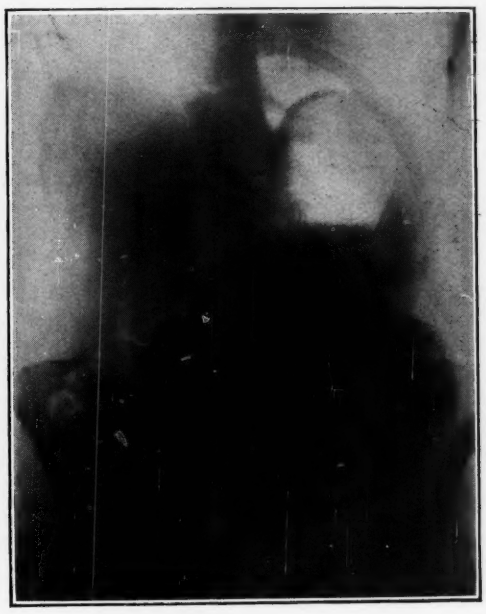


FIG. 346.

This may depend upon the ease with which the gastrohepatic omentum can stretch, and not upon congenital variations in its length, since prolapse of the stomach in association with a mobile ascending colon is but rarely present in children. Prolapse of the stomach and

liver are phenomena that appear comparatively late in the course of these cases, and represent an inevitable reaction to a constant but slowly acting force, the effects of which will be felt earliest along the direct path of action, i.e., the right kidney, duodenum, gall-bladder, and pyloric region of the stomach, and at a later stage in more remote regions, through a secondary distribution of its force to the liver, cardiac end of the stomach, and even the left kidney. A general prolapse of all the viscera of the abdomen can thus be represented as an orderly process which is the natural corollary of the physical stress entailed by the attempt to sustain the weight of the growing ascending colon, and its progressively increasing load, upon so attenuated a structure as a mesentery. Therein, too, lies the explanation of the right kidney being the only mobile kidney in 80 per cent of all mobile kidney cases ; of 15 per cent of the remainder being always bilateral ; and of the inability to refix the right kidney securely to its normal position (Thomson Walker). Surgical operations for that purpose do not deal with the force that has brought about prolapse, and, through the continuation of its action upon the new attachment after operation, prolapse is once more brought about.

GENERAL CLINICAL CHARACTERISTICS.

The cases in this series may be divided into types—'gastric,' 'duodenal,' 'biliary,' 'renal,' and 'right iliac fossa,' according to the character and localization of the pain. But they manifest clinical signs and symptoms which are common to all of them at first, and subsequently become obscured by the appearance of the type pain which in the estimation of the patient relegates his other symptoms to the background. The onset is generally dated by the patients from the age of twenty years or thereabouts, and described by them as the appearance of 'indigestion.' This is no more than a feeling of fullness and discomfort after meals, flatulence, and some lethargy. This indigestion cannot be explained either by indiscretions of diet, bad habits of life, or unsuitable environment, and it is not amenable to any change from one of the supposed causes. The average duration of these symptoms is about six months, and then pain in one or the other region of the abdomen appears for the first time.

During these six months many remedies for 'indigestion' have generally been tried without effect : and with the appearance of pain the patient is no longer content to regard the trouble as being of a trivial nature. Particular emphasis must be laid upon one aspect of 'pain' in these cases : *although it is constant in type and constant in location for the individual case, it is characteristically erratic and irregular in its times of appearance*, both with reference to the number of days on which it may be present, followed by lucid intervals of entirely irregular duration, and to its appearance, disappearance, and reappearance sometimes in the course of a single day. This erratic chronology of the pain is so essential a feature of these cases that it assumes the position of a sign of much diagnostic importance. Granted that the pain has an organic origin, then the factor that produces it must clearly be one that is erratic and variable in its action.

Now the load of the mobile ascending colon, and consequently the strain that it exerts, are most certainly variable. Herein appears to lie the explanation of what is such a marked feature of these cases, namely, the constant inconstancy of the pain. Perhaps, too, this inconstancy affords an explanation of the diagnosis that had already been presented to so many of the patients that they were 'neurotics' or 'neurasthenics.'

Added to the presence of pain, loss of weight has now become a noticeable feature, and profound lassitude has become a disquieting and daily experience. Many of the patients in this series record a loss of from 14 lb. to 28 lb. during the first year of their illness. In the later stages general wasting and tissue degeneration all over the body is one of the grossest features of the condition.

With the exception of 31 cases out of the total series of 180, constipation had never preceded their symptoms ; these 31 had been constipated since childhood, and in those who subsequently became so, constipation did not appear as a rule until after an average of four years' suffering. Constipation in these cases is the result of the final

breaking down of the colon in performing its natural function under mechanical conditions badly suited to it, and cannot be regarded as the cause of symptoms antecedent to it by about four years.

Pregnancy, as might be expected, affords great relief to these patients when the broad plateau of the gravid uterus supports the prolapsed colon. The symptoms generally disappear so completely that the patients are convinced of the soundness of the advice that " if only they could have a baby they would get well " ; their disappointment is bitter at the re-appearance of their old symptoms when the baby is about two months old and their return to active life without the support of a gravid uterus has once more exposed their ascending colon to the unrestricted action of the force of gravity.

On examination of the abdomen, the fullness of the cæcum and ascending colon is easily ascertainable, and in 79 per cent of the cases the right kidney showed variation of position between a gross prolapse and slightly increased mobility and palpability. This

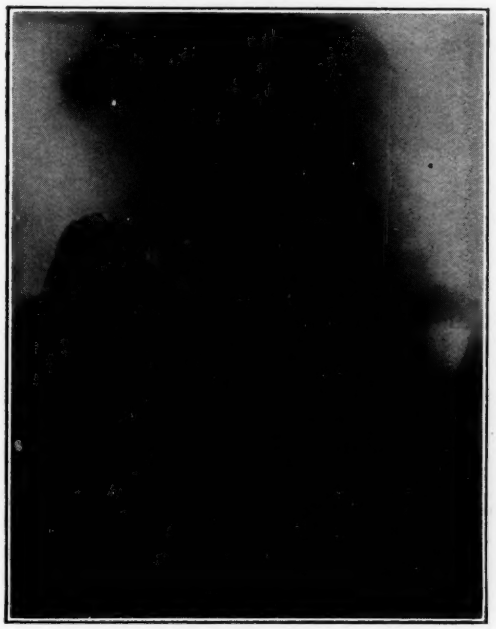


FIG. 347.—Case of *gastric crises* lasting for about two hours, and then sudden relief with the appearance of an intense discomfort in the *right iliac fossa* lasting for many hours. Taken 5 hours after meal, in the erect position, at the end of deep inspiration.

FIG. 348.—Same case as *Fig.* 347, but taken 5 hours after meal in the prone position at the end of deep expiration. A comparison between the levels of the hepatic flexure in the two figures shows the range of excursion of the ascending colon. A complete primitive mesentery was found to be present at the subsequent laparotomy.

is a clinical sign that must be regarded as of more importance in considering the diagnosis of disorders of the alimentary tract than of the urinary tract, when taken in conjunction with the observation that although a mobile kidney was present in so large a proportion of the patients, no symptoms were present that could possibly be attributed to it. A few of the patients had renal crises in addition to their other symptoms, and these were relieved by operation upon the colon without any surgical procedure being performed on the kidney.

X-ray Examination. — Radiographic records of barium or bismuth meals showed an obvious malposition of the ascending colon as judged by the position of the hepatic flexure in all cases. As a rule this was as low as the level of the hinder margin of the iliac crest, and in many cases was much below even that level in the upright position (*Figs.* 344 to 357). The range of excursion of the ascending colon was estimated by comparison

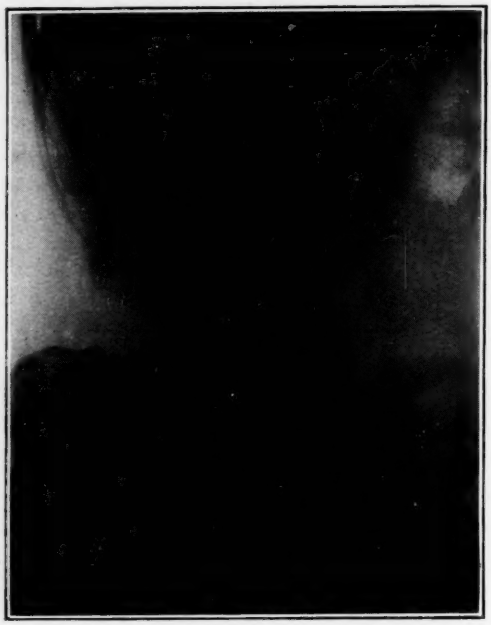


FIG. 349 (*Case* 8).—*Biliary crises.* Here the hepatic flexure lies entirely below the level of the iliac crest. The gall-bladder seemed to be perfectly healthy at the subsequent laparotomy, and there were no calculi present, but a band of peritoneum passed between the under aspect of the gall-bladder over the duodenum to the ascending colon in the region of the hepatic flexure. Taken in the upright position 24 hours after the meal.

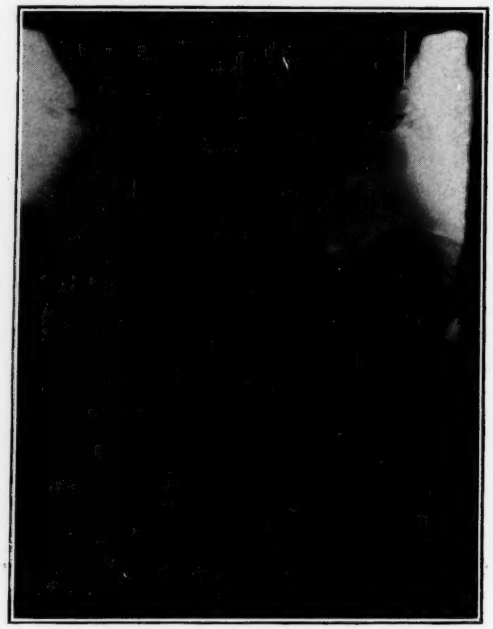


Fig 350.—Case of *multiple renal crises.* Here the hepatic flexure lies entirely below the level of the iliac crest. Taken in the prone position 8 hours after the meal (Case 13).

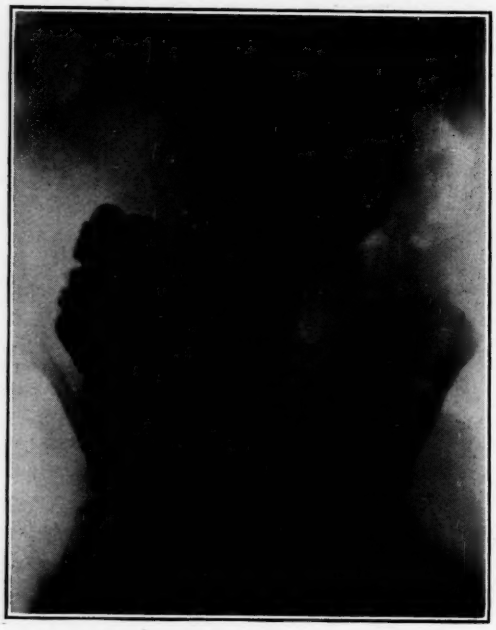


FIG. 351.—Type of appearance as seen in *a child, age* 11 *years.* Taken eight hours after barium meal at end of inspiration in erect position. Stomach still contains food, and hepatic flexure is seen to be just above the level of the iliac crest. Patient had suffered for several years from attacks of vague discomfort in right iliac fossa, accompanied by nausea and fever lasting for several days.

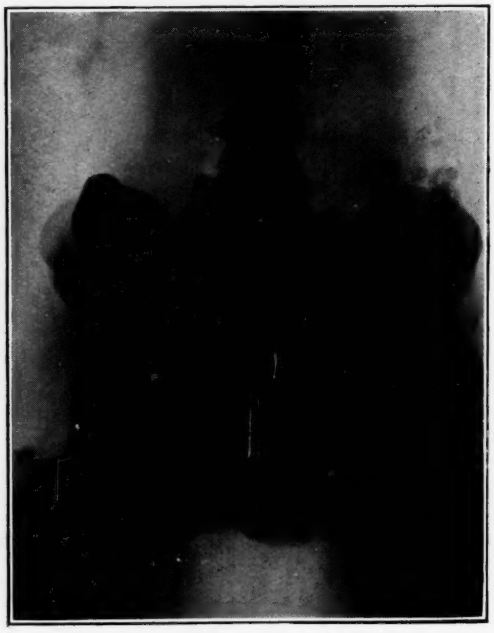


FIG. 352.—*Duodenal type*, taken 2¼ hours after meal, in erect position at end of deep inspiration. No ulcer found at subsequent operation, but a complete primitive mesentery to the ascending colon. The hepatic flexure is well below the level of the iliac crest.

of plates taken in the lying-down position at the end of expiration, and in the upright position at the end of inspiration, thus showing the highest and lowest points respectively occupied by the hepatic flexure (*Figs.* 347 and 348). The low level of the greater curvature

FIG. 353.

FIG. 354.

FIG. 353.—*Duodenal type*, taken 8 hours after the meal, in the prone position, at the end of expiration. The hepatic flexure is just above the level of the iliac crest, and the steep ascent to the splenic flexure is also shown on the left side. No ulcer was found at the subsequent operation, and a complete primitive mesentery to the ascending colon was present.

FIG. 354.—*Gastric type* with a history of one *renal crisis*. Taken 4 hours after a meal, in the erect position, at the end of deep inspiration. The hepatic flexure can be seen well below the level of the iliac crest.

FIG. 355 (*Case* 22).—*Duodenal ulcer*, taken 4 hours after a meal, in the erect position, at the end of inspiration. Ulcer in 1st part of duodenum and a complete mesentery to the ascending colon. The hepatic flexure lies at the level of the iliac crest, and no delay in the escape of the meal from the stomach, nor irregularity of the duodenal cap, was detected radiographically. The patient had had one severe attack of melæna.

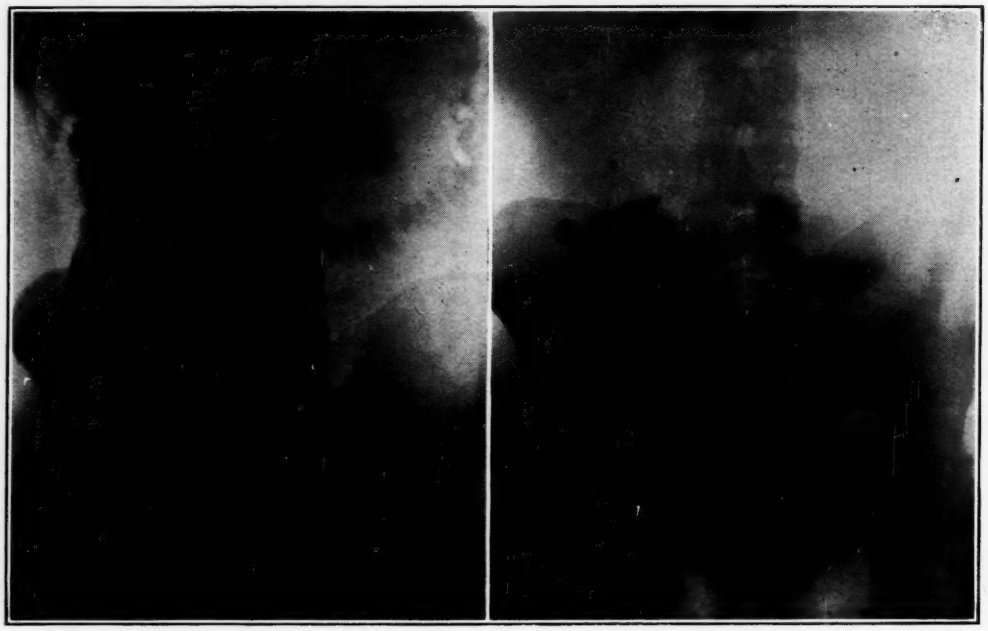
FIG. 355.

of the stomach was easily seen, as was also the steep ascent of the left half of the transverse colon to the fixed splenic flexure in some of the cases (*Fig.* 353).

Variations in the outline of the stomach and duodenal cap were occasionally recorded, but they were not correlated with any detectable organic lesion at the subsequent operation,

and no deductions with regard to diagnosis were made from their presence or absence. As observations they were peculiarly worthless, for in those cases where an irregularity of outline was recorded, no ulcer was detected in any case at the subsequent operation, and in the few patients with known ulcer who were well enough to be submitted to radiographic examination beforehand, no irregularity was detected, although the ulcer was easily found at the operation. Finally, a few cases of ulcer were far too ill to be subjected to radiographic examination beforehand, but the diagnosis of ulcer *and mobile ascending colon*, on clinical signs and symptoms only, was always confirmed at the subsequent operation.

Delay in the passage of the opaque meal from the stomach, ileum, and ascending colon was generally present, but when once the splenic flexure was passed, little or no delay was noticeable. The rate of passage of a foreign substance through the alimentary tract appears to the writer to be but a poor foundation upon which to build a diagnosis of the pathological disorder of such cases as these. The mode of preparation of the patient varies with the individual radiographer—some of them administer a dose of castor oil overnight, and the determination of its power of acceleration is entirely beyond control.



FIGS. 356 and 357.—Radiograms to show the range of excursion of the ascending colon from the prone to the upright position. Taken 7¼ hours after a barium meal. Food still present in stomach and ileum. Patient was a man 27 years of age, with a history of erratic gastric pains for six years, with three attacks of hæmatemesis during that period.

Many of the patients are exceedingly nervous at the ordeal they have to undergo, some of them are nauseated by the particular preparation they have to swallow, many of them have their ordinary habits upset by the frequent visits to the radiographer to enable a series of plates to be taken; in fact, a normal healthy person may have many temporary reasons for the inhibition or acceleration of the passage of the meal. The character of the drug—bismuth or barium—must also be taken into account. Finally, the delay cannot be demonstrated at the time of operation, and assuredly not the reason for it, unless some gross obstruction of a portion of the alimentary tract is present. Many of the patients in this series who had already had the appendix removed—altogether 68 in number—before coming into the writer's hands, had been assured that the delay of the bismuth in the ileum and stomach was due to the irritability of the diseased appendix—'controlling appendix' was the term used—and that this would disappear after the appendix had been removed.

Therefore the rate of passage of bismuth or barium has not been used as a basis for diagnosis in these cases. It has been observed as an associated phenomenon, the value of which for diagnostic purposes is very limited indeed; and radiography has been used primarily to demonstrate the presence of a prolapsed mobile ascending colon—a simple determination that admits of no dispute, and one that is confirmed beyond all peradventure at the subsequent laparotomy. So far the premises have been simplified, and the fact of their existence can be proved by any observer.

TYPES.

These are recognized only when pain has made its appearance.

Gastric Type.—Pain is experienced in the stomach area somewhere along the left costal margin and ensiform process of the sternum. It comes on very soon after food, within ten minutes to three-quarters of an hour after a meal has been taken. But it does not come on after every meal, and it does not necessarily appear every day; sometimes it may come on just before a meal, and though as a rule the taking of food then makes it worse, it sometimes makes it better. It is generally described as being 'nagging' or 'boring' in character, and may radiate through to the back. Localized tenderness can often be detected by the patient over the point of maximum intensity. Thus the location and character of the pain are constant for each patient of this type, but the times and seasons of its appearance are variable. It does not necessarily appear after every meal in any given day, nor does it appear every day in an unbroken sequence. Sometimes there may be an interval of a few weeks between the attacks of pain, sometimes a few days, sometimes only a few hours; sometimes there may be many weeks of pain with only a brief lucid interval of a few hours' duration. It is unaffected by the quality of the food taken. Most of the patients of this series, after several years of dieting under skilled advice, had thrown all restrictions away, and unhesitatingly affirmed that they had derived no benefit from model dietaries, and were no worse when they ate what they liked.

Several of the patients had been regarded as the victims of gastric ulcer, a diagnosis that is negatived by the erratic behaviour of the pain. When an ulcer has once formed, pain is constant in its times of appearance; it always comes on after food, it is always made worse by food, and there are no lucid intervals. But when a pain is caused by a strain that is variable in its intensity, then it will only make its appearance when the strain is effective up to the point of producing pain, that effectiveness being determined by the load which the dropped colon is carrying. None of these patients with inconstant gastric pain have been found at laparotomy to have had structural disease of the stomach; and all of them have had a mobile ascending colon; whilst in the patients in whom ulcers were known to be present before operation (*vide* details of ulcer cases), and a mobile ascending colon was always found at operation, the pain had been constant in its appearance immediately after taking food, and so severe as to make each meal an intolerable experience.

This constant response to a stimulus is a character of ulcers generally in other parts of the body, and the gastric ulcer appears to be no exception. Moreover these patients (i.e., those with gastric ulcers) had obviously benefited from restrictions of diet for brief periods; the restrictions were often self-imposed, but since they had no direct healing action upon the original ulcer, the time had come for most of them when even the taking of Benger's food was an ordeal they hesitated to face. The inconstant gastric pain is not due to an ulcer, but to a variable strain produced by a variable load in the offending segment of gut.

Nausea is sometimes present, but actual vomiting appears, from the histories of the patients, to be of very rare occurrence. So clear does the differentiation seem to be, by means of the histories, between the pain from the strain on the stomach of a mobile colon and from a gastric ulcer, that occult blood has never been searched for, and this omission has not been penalized by the unexpected discovery of an ulcer at the subsequent laparotomy. All the cases of ulcer had had gross hæmatemesis or melæna, or both. The need for the test appears to arise from an inadequate evaluation of the patient's

symptoms, with the apprehension that a laparotomy may be performed and no ulcer be found. An operation should be performed with the certainty that no ulcer will be found, and the high expectation that benefit will accrue from it.

Duodenal Type.—Pain is experienced in the right hypochondrium, and is characteristically a 'hunger pain' that appears about three hours after food and is relieved by the further taking of food. But once again, whilst its character and location are constant, its times of appearance and response to the taking of food are constantly inconstant. It is not necessarily a daily experience, nor if it appears before one meal in the day does it always recur before each subsequent meal. As a rule pain is relieved by the taking of food, but sometimes it is not relieved, and sometimes it is made worse. When the strain of the mobile ascending colon falls with maximum intensity upon the second part of the duodenum—the site that is most liable to ulceration—taking of food can clearly have one of two effects upon the load in the ascending colon. Either the wave of peristalsis initiated by the next meal shifts the load into the transverse colon, and so relieves the strain upon the duodenum, or the meal merely silts up in the ascending colon, and adds to its load, thus failing to relieve, or even intensifying, the pain. As a rule the former happens, and so the phenomenon of a 'hunger pain'—that is, a pain relieved by food—is established. Alternatively the pain is made worse—a rarer event—in an easily explicable manner, when the food merely adds to the load of the ascending colon. Further, a new light appears to have been thrown upon the origin of 'hunger pains,' which it must be emphasized have been studied in all these patients in whom no duodenal ulcer could be detected at the subsequent operation, in entire accordance with one's expectations.

The time of appearance of a 'hunger pain,' coincides as a rule with a time when most of the previous meal will have arrived at the ascending colon—that is, within about three to four hours after feeding. When pain has been established, it may as a rule be relieved by inducing further peristalsis in the alimentary tract by taking food—a method of relief which sometimes quite significantly fails, as has already been explained. Hence the 'hunger pain' is really a 'food pain' determined by the arrival of food at a portion of the alimentary tract where it can exert a nocuous strain, exhibiting a variability that would be expected from a variable strain, and responding in an erratic manner to a method of relief, that is not strictly controlled, for augmenting or diminishing the strain. The difficulty of explaining a 'hunger pain' by the presence of a duodenal ulcer—even if the inability to detect an ulcer in all these cases was assumed to be a personal failure—is that a duodenal ulcer, in being soothed by active movement and arrival of foreign material in the duodenum—since the food must get out of the stomach some time—is made to exhibit characteristics which ulcers elsewhere do not display. On the contrary, in the few cases of actual duodenal ulcer in this series, the patients have been most emphatic in their statements that after a preliminary period of many months and sometimes years of intermittent 'hunger pains,' their pain in the right hypochondrium has now been constant for many months, and is always made worse by food. They can recognize very vividly for themselves the change between the pain that was an erratic visitor soothed by food, and one that has now become a constant companion from which no relief can be found.

The knowledge of the association between 'hunger pains' and a duodenal lesion is a debt that surgery owes to Moynihan, and further elucidation of its details can only be regarded as an amplification of his pioneer work, and as a grateful recognition of the stimulus that he has provided to further endeavour.

Biliary Type.—Several patients have given a history of attacks suggestive of gall-stones—some have received expectant treatment with the diagnosis already provided by their medical attendant ; but only those cases are included under this heading in which the actual crisis has been witnessed by the writer. The crisis is very severe, and characteristic of obstruction to the outlet of the gall-bladder, either at the neck or at the cystic duct. The onset is sudden, and intense pain is experienced in the region of the gall-bladder, leading to vomiting and collapse. The upper segment of the right rectus is too rigid for any underlying structure to be recognized by palpation. The attack may last for twelve to thirty-six hours, and when it has passed off, with relaxation of the rectus, a tender,

swollen gall-bladder can easily be felt. This shrinks to normal size in a few days' time. Jaundice, except for an occasional faint tinging of the conjunctiva, does not appear ; the stools contain plenty of pigment, and bile does not appear in the urine. The attacks are non-febrile as a rule, although in one case elevation of temperature occurred for forty-eight hours. At the subsequent laparotomy, performed generally within a fortnight after the crisis, a healthy gall-bladder without calculi was present in each patient, and a mobile ascending colon with its primitive mesentery. But an additional feature of great interest, and needing further investigation, was present in some of the cases. The gall-bladder was attached either to the second part of the duodenum, or to the hepatic flexure of the colon, by a band of membrane passing from these structures to the ventral surface of the gall-bladder and cystic duct over most of its length. This membrane is so constant in its attachment along a line to the ventral surface of the gall-bladder that it does not appear to be of inflammatory origin. It has well-defined lateral surfaces with small blood-vessels upon them, a free edge towards the fundus of the gall-bladder, and a broad base that fuses with the anterior layer of the gastrohepatic omentum (*Fig.* 358). On cutting the membrane to free the gall-bladder, two edges with cellular tissue between them are left along the line of section. Such features are suggestive of a persistence of a small portion of the primitive anterior mesogastrium, between the layers of which the gall-bladder develops, rather than of adhesions of an inflammatory origin, which would in all probability show a more haphazard distribution. The point needs further investigation ; but of whatever origin, it is clearly one medium whereby the strain from a sagging ascending colon can be transferred direct to the gall-bladder and cystic duct. The other medium is the gastrohepatic omentum, which, sagging under the weight of the colon, bends the cystic duct at an abrupt angle to the neck of the gall-bladder. If complete obstruction is caused through either of these means, then an acute biliary crisis follows. But it is conceivable that only partial obstruction may be caused in a number of cases, not sufficiently severe to produce a crisis, but sufficient to add the element of stagnation to the circulation of the bile in the gall-bladder, so that the arrival of attenuated bacteria results in the formation of gall-stones. A few patients in this series, with a mobile ascending colon but no symptoms suggestive of a lesion of the biliary apparatus, were found to have gall-stones also at the operation ; in their case a silent formation of gall-stones had occurred without incidental complete blocking of the cystic duct either from within or from without. Since the gall-bladders of all human beings, and especially all typhoid patients, are subjected to the influence of bacteria arriving from the alimentary tract from childhood upwards, whilst gall-stones are not formed in all of them, it is possible that stagnation is the inconstant factor, which, in its turn, depends upon the inconstant survival of the mesentery of the ascending colon.

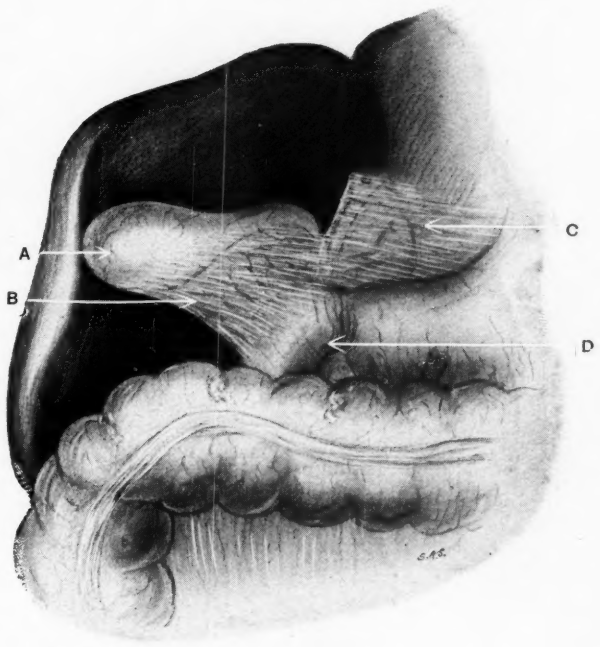


FIG. 358.—Shows vestigial band (B) running between the ventral aspect of the gall-bladder (A) and cystic duct and the duodenum (D). It has a free margin below, while mesially its fibres blend with the anterior surface of the gastrohepatic omentum (C). Upon its surface small blood-vessels can be seen. The liver has been raised and rotated to expose the biliary apparatus.

Cases of Biliary Crisis.—

Case 1.—K. H. S., male, age 43. Ten years' gross dyspepsia. Appendix removed five years previously. May 14, 1912, acute biliary crisis lasting forty-eight hours. May 24, 1912, laparotomy: Gall-bladder quite healthy in appearance; no calculi; complete mesentery to ascending colon, fixed in usual way. Heard from patient April, 1919: "No attacks since operation: dyspepsia much better, but not quite cured."

Case 2.—R. W., male, age 34. Five years' dyspepsia; gave a history of acute attacks of pain lasting several hours; typical crisis seen Feb. 5, 1914. Appendix removed three years previously. Feb. 20, 1914, laparotomy: Healthy gall-bladder with band running to hepatic flexure; no calculi; complete mesentery to ascending colon; fixed in usual way. Seen May, 1918: said he was in perfect health, and had put on 2 stones in weight.

Case 3.—H. W., male, age 31. History of dyspepsia and acute attacks of pain for two years. Typical biliary crisis seen on Feb. 21, 1916. Laparotomy, March 16, 1916: Healthy gall-bladder, with band from ventral surface to second part of duodenum; complete true and pseudo-mesentery to ascending colon; gut fixed in usual way; small, apparently healthy appendix removed. Seen two years later: no further attacks, digestion good, and had put on 1½ stones in weight.

Case 4.—T. H. N., male, age 25. "Treated for gall-stones for two years and then invalided back from front." Appendix removed five years previously. Biliary crisis seen Aug. 11, 1916. Laparotomy, Sept. 1, 1916: Healthy gall-bladder; no calculi; no band; mesentery to ascending colon; gut fixed in usual way. Quite well a year later, and had returned to active service at the front.

Case 5.—R. T. K., male, age 26. Dyspepsia four years, no previous crisis: seen in typical biliary crisis, Feb. 15, 1917. Laparotomy, Feb. 27, 1917: Similar conditions and operation as *Case* 3. Seen seven months later, when he reported as quite well. Returned to active service at front, and no further history obtainable.

Case 6.—R. C., male, age 28. Always had dyspepsia, and several attacks of severe pain. Biliary crisis seen on March 28, 1917. Gall-bladder healthy; no calculi; no bands; mesentery to upper half of ascending colon and pseudo-mesentery to lower half, fixed in usual way; healthy-looking appendix removed. Reported "quite well," Sept. 28, 1918.

Case 7.—T. W., male, age 31. "Suffered from gall-stones for several years." Biliary crisis, April 23, 1917. Laparotomy, May 7, 1917: Condition and operation similar to *Case* 3; healthy gall-bladder; no calculi. Quite well eight months later, and returned to active service at the front.

Case 8.—R. A., male, age 23. "Acute attacks of abdominal pain since 6 years of age. Under continuous treatment since 1910." Joined army in 1914, and has been on active service abroad; invalided home three times since 1916; admitted under care of writer Oct. 5, 1918, with diagnosis of gall-stones. Several biliary crises witnessed after admission. Laparotomy performed Oct. 28, 1918: Healthy gall-bladder with band running from ventral surface to hepatic flexure; no calculi; true primitive mesentery to the upper three-fourths of ascending colon; gut fixed in usual way; small healthy appendix removed. Reported July, 1919, "in perfect health and no further attacks. Could eat anything." (*Fig.* 349).

Case 9.—G. P. D., female, age 38. Ten years' dyspepsia, with three acute attacks of pain in right hypochondrium lasting about eight hours. Gall-bladder palpable when seen Nov. 13, 1918. Laparotomy, Jan. 15, 1919: Healthy gall-bladder; no calculi; band from ventral surface to hepatic flexure, complete mesentery to ascending colon; gut fixed in the usual way.

Case 10.—F. A. G., male, age 35. Dyspepsia and constipation for some years, bad headaches, subacute biliary crisis lasting for ten days. Feb. 1919, gall-bladder palpable. Appendix removed some years ago. Laparotomy, March 3, 1919: Healthy gall-bladder; no calculi; band from neck of gall-bladder to second part of duodenum; complete mesentery to ascending colon; gut fixed in usual way. Has been free from constipation, headache, and pain. Had gained 6 lb. in weight by September, 1919.

Renal Type.—In addition to their general symptoms of indigestion, these patients experience renal crises of great severity. Several have given a history that "they were suspected of having a stone in the kidney," but the only four included amongst this type are those in whom the crises were witnessed by the writer. They were so severe as to produce collapse and vomiting, whilst the right kidney was swollen and tender. (One patient had 11 crises under observation.) When the attack is over, the kidney rapidly shrinks in size, but there is no increased output of urine, and the normal quantity that is subsequently passed is found under the microscope to contain a few red blood-corpuscles. Apparently a partial occlusion of the renal vein is brought about by the drag of the colon during the attack; and it is of interest to note that, in the case of multiple crises, the attack

could be cut short by the administration of a high glycerin enema, accompanied by a hypodermic injection of pituitrin. The usual routine examination for a renal or ureteric calculus was made beforehand in all these patients, with negative results ; their urine was normal in composition in the intervals of their attacks ; and at the subsequent laparotomy it was not possible to detect anything wrong with the kidney or ureter in any of them. All of them had a mobile ascending colon.

Case 11.—A. S., female, age 36. Dyspepsia and vague abdominal pains for some years, appendix removed three years previously. Several attacks of pain in right lumbar region, followed by passage of blood-stained urine. Sept. 8, 1916, typical right renal crises. Sept. 15, 1916, laparotomy : Complete mesentery to ascending colon ; gut fixed in usual way. Reported in January, 1919, quite well and no attacks since operation.

Case 12.—I. G., female, age 39. Five years' history of abdominal pains, chronic discomfort in right iliac fossa, with occasional acute pains in right lumbar region. Three years' very bad constipation, and lived on drugs. March 27, 1917, renal crises with swollen right kidney. April 7, 1917, laparotomy : Complete mesentery to ascending colon ; gut fixed in usual way ; small healthy-looking appendix removed. June 30, 1919 : " Has been quite well ever since operation, has no pains, and bowels act regularly every day without any medicine."

Case 13.—T. H., male, age 38. Many years of dyspepsia and attacks of acute pain in kidney region. Appendix removed seven years ago, no relief ; another laparotomy two years later to " undo a kink," but no relief. Admitted to hospital with gunshot wound of arm, Aug. 28, 1918. Renal crises of right kidney began early in September, and eleven were witnessed up to Oct. 7, 1919, when a laparotomy was performed. Mesentery to whole of ascending colon ; gut fixed in usual way. Was quite well and had had no further attacks when last seen on March 12, 1919. (*Fig.* 350).

Case 14.—H. J., female, age 36. Had been treated for renal crises and gross dyspepsia for fifteen years ; constipation has come on latterly ; much malnutrition. Appendix removed some years ago, but with no benefit. Laparotomy June 1, 1919 : Mesentery and pseudo-mesentery to whole of ascending colon ; gut fixed in usual way. Dec. 1919 : " Has had no crises since operation, and bowels act daily without drugs."

Right Iliac Fossa Type.—In these cases chronic pain in the right iliac fossa is the salient feature, added to the usual dyspeptic symptoms. It is not focal, and has not the severity of the type pains. It may remain present for several days, and is often described as a severe discomfort experienced diffusely over the right iliac fossa. In some patients it is present in addition to the type pains, and is then considered a trivial disability in contrast with the type pain. It appears to be a sensation experienced from a chronically over-distended segment of gut, to which the element of dragging is added. Several of these patients also give histories of attacks of mucous colitis ; but in one patient the operation on the colon was deliberately undertaken for the relief of persistent colitis of seven years' duration.

Case 15.—A. F., doctor's wife, age 36. Mucous colitis for seven years : appendix removed three and a half years ago, but no improvement followed. Is now a chronic invalid, and steadily getting worse in spite of all forms of treatment tried during the last four years. Laparotomy, Feb. 2, 1918 : Complete primitive mesentery to ascending colon ; gut fixed in usual way. Sept. 22, 1919 : The doctor reports that " his wife is definitely better, is plumper and not so poisoned ; was free from colitis for nine months, but has had one or two slight attacks subsequently."

In the other patients, in whom colitis was not continuous as in this one, but appeared as occasional attacks of about three weeks' duration, there has been no recurrence of the attacks following the operation on the colon. On the other hand, it must be noted in connection with this difficult disorder that in one patient, not included in this series, who had been suffering for a year from a very grave bloody colitis, the ascending colon was not fixed at the operation deliberately undertaken for that purpose ; and the patient made a complete recovery after the removal of the appendix and the fixation of the omentum to the abdominal wall to straighten out the hepatic flexure (Coffey's hammock operation).

Children.—The condition is generally regarded either as 'chronic appendicitis' or merely as 'dyspepsia' in children. Pain, not very severe, which often lasts for several days, is complained of in the region of the right iliac fossa. It is intermittent in character, and examination of the abdomen seldom reveals any obvious explanation of its presence.

There is no real tenderness, no muscular rigidity, but a full ascending colon can be detected when carefully sought for. Vomiting seldom occurs, but nausea and lack of appetite are invariably present. There is often high fever, with a quickened pulse-rate, and an abundance of acetone in the urine. The patient is clearly very ill, and the absence of guiding signs, in the region where the symptoms are experienced, is a source of anxiety that may lead to a panic removal of a healthy appendix. In the absence of tenderness, rigidity, and mass formation, no anxiety need be felt on this score ; whilst the palpation of the outline of the over-distended ascending colon is sufficient to enable a diagnosis to be made. This can be subsequently confirmed, when the attack is over, by an *x*-ray examination (*Fig.* 351). The illness often passes off with a slight attack of diarrhœa. The persistence of a mesentery to the ascending colon has been confirmed at subsequent operation in more than 100 children under twelve years of age, in whom, rather as a diagnosis of despair than a profound mental conviction on the part of their medical advisers, who had witnessed several attacks in each of them, appendicular mischief was said to be present. In 7 of them disease of the appendix was also found at the operation, but in the remainder the appendix appeared to be healthy in every respect. These operations in children have not been included in the total series of 180 cases which are under review in this article. It has not been possible, under war conditions, to follow them up with sufficient precision to justify at present any exact conclusions. Several of them have been seen incidentally at long periods after the operation, and their condition was most satisfactory, but many mothers showed a reluctance to continue bringing their children to hospital merely for the purpose of inspection when they were apparently well.

Between the years 1909–13 the constancy of the presence of a mesentery to the ascending colon in several cases of bacilluria in children (*B. coli* infection) was noted, and laparotomies were performed upon 18 of them. All of them were bad cases that had made no improvement under months and sometimes years of many forms of medical treatment. A dramatic series of cures followed the fixation of the colon in 12 cases, all of them being kept under observation for a year after operation, and all of them showing, as evidence of their cure, complete sterility of twelve consecutive specimens of urine taken at intervals of a month ; this seemed a more severe and more reliable test than the mere disappearance of their symptoms, which was an obvious accompaniment to their improvement. But amongst the last 6 cases no improvement at all followed operation, and no explanation of the failure was found. This was so disappointing that for cases of bacilluria only, the writer abandoned the operation, temporarily at any rate, whilst the significance of the mobile ascending colon was being worked out in other directions.

CASES OF GASTRIC AND DUODENAL ULCERS.

If the mode of formation of these ulcers has been rightly assumed, then not only should a mobile ascending colon be present in all cases, but also fixation of the colon should be followed by the healing of the ulcer in a fair number of cases. Sometimes a very chronic ulcer in any part of the body will not heal when the mechanical factor responsible for its formation has been removed, on account of pathological changes in the immediate neighbourhood of the ulcer too well known to be recapitulated here.

Nevertheless, in the few ulcers that have come under the writer's experience during this period (with the exception of perforated ulcers, in which the operation was not attempted), the ascending colon has always been mobile, and the ulcers have healed when the operation of fixation has been performed. Four gastric ulcers and six duodenal ulcers have been submitted to operation and the colon fixed : one patient with gastric ulcer died from hæmatemesis before an operation could be performed, and at the post-mortem examination an ulcer and a complete primitive mesentery to the ascending colon were present ; on a patient with gastric carcinoma, apparently originating in a large saddle-back ulcer, an exploratory laparotomy was done, and the whole of the ascending colon was lifted out on to the towels ; but the condition of the stomach and the patient were too hopeless to permit of any further operative procedure being done.

As has already been mentioned, actual ulcers have been very rare in this series of cases, whilst type pains of gastric and duodenal distribution have been very common. When an ulcer was present it was so gross and obvious a lesion, both to the operator and his assistants, that it is difficult to suppose they were overlooked in other cases.

Gastric Ulcers.—

Case 16.—E. S., female, age 31. One attack of hæmatemesis June, 1915. Gastric pains five years. Appendicectomy four years previously, dyspepsia many years. Laparotomy, Aug. 4, 1915: Indurated ulcer size of a shilling on anterior surface of stomach just to left of pylorus; complete primitive mesentery to ascending colon; colon fixed in usual way. Made a very rapid recovery, and reported quite well on May 30, 1918.

Case 17.—D. H., female, age 36. Had been treated for four years in different hospitals for gastric ulcer, during which period two grave attacks of hæmatemesis had occurred; appendix removed seven years previously. Admitted, blanched from third attack of hæmatemesis, to Hampstead General Hospital on June 25, 1917. Very ill, and treated by conservative measures until Aug. 9, 1917, when laparotomy was performed: Large ulcer, size of a half-crown, on anterior surface and upper border of stomach to left of pylorus; complete mesentery to ascending colon, which was fixed in usual way. Up to date of operation had not been able to take anything more solid than Benger's food, and then with severe pain; ate fish on tenth day, and meat on fourteenth day, after operation. Anæmia rapidly improved, and she returned to work four months after operation. Reported herself quite well in June, 1919.

Case 18.—J. D., female, age 27. Dyspepsia several years, gastric pain one year, severe melæna on several occasions, and hæmatemesis on July 17, 1917, when she was admitted to Hampstead General Hospital. Laparotomy, Aug. 25, 1917: Two ulcers present about size of a shilling and a sixpence respectively, on anterior wall of stomach and duodenum on either side of pylorus; true mesentery to lower half of ascending colon and pseudo-mesentery to upper half; colon fixed in usual way after apparently healthy appendix had been removed. Uninterrupted recovery, and reported on June 15, 1919, to say that she was in perfect health.

Case 19.—A. H., female, age 29. Dyspepsia for some years, constant gastric pain for six months, even with fluid diet. Laparotomy, Oct. 16, 1918: Ulcer size of a sixpence on anterior wall of stomach close to lower border, about 1½ inches from pylorus; complete mesentery to ascending colon. Fixed colon in usual way, and small, apparently healthy, appendix removed. Uninterrupted recovery. Wrote Aug. 6, 1919, to say that she had "never been better in her life."

Duodenal Ulcers.—

Case 20.—D. B., female, age 27. Dyspepsia one year, hunger pains three months, with two attacks of melæna lasting about twelve days. Laparotomy, Aug. 11, 1917: Complete true mesentery to whole of ascending colon. Ulcer size of sixpence in second part of duodenum, just above hepatic flexure. Colon fixed in usual way, and healthy appendix removed. Writes Aug. 10, 1919, to say that "she has been in perfect health ever since operation."

Case 21.—A. H., male, age 47. Dyspepsia for some years. Hunger pains six months. One attack of melæna lasting for ten days in January, 1918. Laparotomy, March 20, 1918: Large ulcer in second part of duodenum, just above hepatic flexure; complete true mesentery to ascending colon. Colon fixed in usual way, and small healthy appendix removed. Reports September, 1919, that "he is quite sound."

Case 22.—E. H., female, age 38. Many years' treatment for dyspepsia and chronic pain. Three attacks of melæna during past five years. Appendix removed seven years ago. Laparotomy May 11, 1918: Large ulcer in second part of duodenum; true mesentery to lower two-thirds of ascending colon, and pseudo-mesentery to upper one-third. Colon fixed in usual way. September, 1919, still has slight dyspepsia, but no pain, and considers herself "to be better than she has been for many years."

Case 23.—E. S., female, age 26. Dyspepsia and hunger pains on and off for five years, Appendix removed two years ago, no relief. Laparotomy, March 2, 1919: Small ulcer in second part of duodenum; complete mesentery to ascending colon; gut fixed in usual way. August, 1919, "so far very well."

Case 24.—A. A., female, age 31. Many years' dyspepsia and pain, one attack of melæna lasting seven days. Appendix removed three years ago; no better. Laparotomy, May 2, 1919: Ulcer size of a sixpence in first part of duodenum; complete mesentery to ascending colon. Colon fixed in usual way. September, 1919, has put on 10 lb. since operation and is very well.

Case 25.—F. L. C., female, age 29. Dyspepsia and chronic pain for last nine months, during which period had two attacks of melæna and one of hæmatemesis. Laparotomy, May 22, 1919: Ulcer in first part of duodenum just to right of pylorus; complete mesentery to ascending colon; gut fixed in usual way, and small healthy-looking appendix removed. Nov. 1919: "Has been quite free from pain, and only has slight flatulence occasionally."

FORMATION OF A GASTRIC ULCER IN A PATIENT IN WHOM A MOBILE ASCENDING COLON HAD BEEN PREVIOUSLY DEMONSTRATED.

This case is of interest as an intermediate type between those with gastric pains and those with gastric ulcers, and illustrates the transition from the former to the latter whilst the patient was under observation and known to be the possessor of a complete mesentery to the ascending colon.

An operation was performed for the relief of the gastric pain before the significance of the persistent mesentery as a causal agent had been appreciated, and although its presence was demonstrated at the operation, fixation of the colon was not performed. The pains returned a few months later, and after they had persisted for three to four years, two grave attacks of hæmatemesis occurred, in one of which the patient nearly died.

Mrs. C., age 28, seen with Dr. McD. Ellis on Dec. 19, 1912. A history of dyspepsia of a gastric type for a few years had been interrupted six months previously by an attack of biliary colic. Laparotomy advised and performed on Jan. 17, 1913: No structural disease found, with the exception of adhesions surrounding an enlarged appendix, and bands between the gall-bladder and the hepatic flexure. The whole of the ascending colon was lifted out on to the towels, and the persistence of its primitive mesentery demonstrated. The appendix was removed, the bands between the gall-bladder and hepatic flexure were divided, and the great omentum was then attached transversely across the abdominal wall (Coffey's hammock operation). The symptoms returned a few months after the operation, and continued to get worse in spite of all forms of expectant treatment. In 1917 the patient had two very severe attacks of hæmatemesis, one of which was nearly fatal. She refused further operative treatment.

OPERATIVE TECHNIQUE.

A sand-bag is placed under the lower part of the thorax, as for gall-bladder operations, to render easier the access to the hepatic flexure. The lower two-thirds of the right rectus

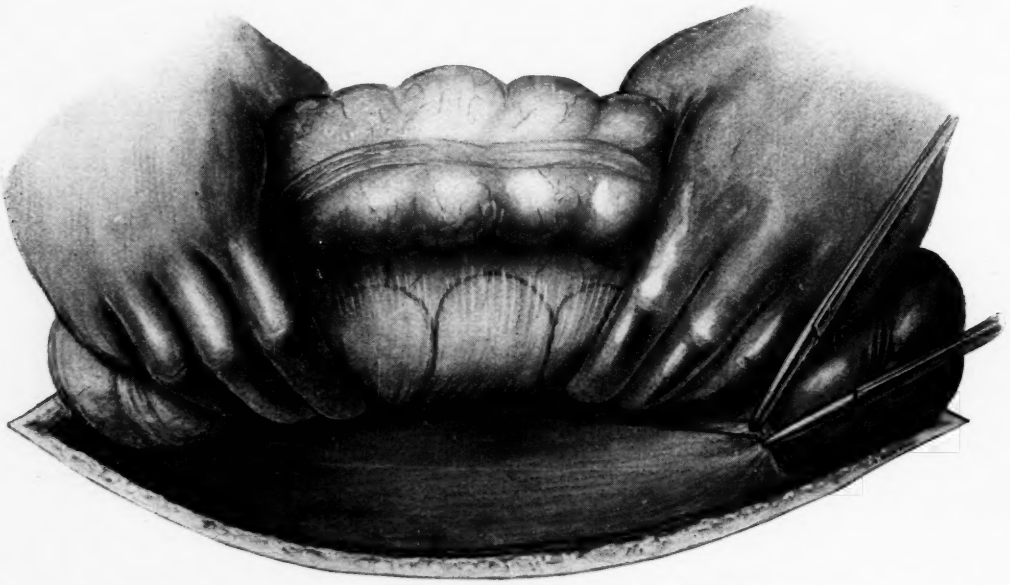


FIG. 359.—The incision of the fascia covering the posterior abdominal wall is shown by the dark line running from the level of the pelvic brim to the hepatic flexure close to the root of the primitive mesentery.

is turned inwards, and the nerves are mobilized at the points where they pierce the sheath and the muscle respectively. As a rule it is then possible to preserve them intact, although the eleventh thoracic nerve is sometimes snapped by the displaced colon. The abdominal and pelvic contents are then carefully palpated and inspected, so that a view of most of

the stomach, the duodenum, the biliary apparatus, the ileum and appendix, the ovaries and tubes, and finally the colon itself, is obtained. The region where the iliac colon passes over the left side of the pelvic brim is palpated to exclude any bands of thickening in its mesentery. The ascending colon, from cæcum to hepatic flexure, is then lifted out on to the towels (*Figs.* 338, 339, and 340), and at this stage the main lines of strain upon the stomach, duodenum, and gall-bladder can be demonstrated easily by gentle tugs upon the hepatic flexure (*Fig.* 342). The mobility of the second part of the duodenum is tested by lifting it up between the finger and thumb, when, more particularly in cases of hunger pains, it can be brought up to the level of the incision in the skin (*Fig.* 341). The appendix is removed if present, bands between the gall-bladder and colon divided (*Fig.* 358), and rare bands, as those causing an ileal kink and those occasionally found at the brim of the pelvis on the left side, are similarly dealt with. The peritoneum and fascia are then incised just above the brim of the pelvis on the right side, and the incision is carried upwards parallel to the line of the colon and as close as possible to the root of its mesentery

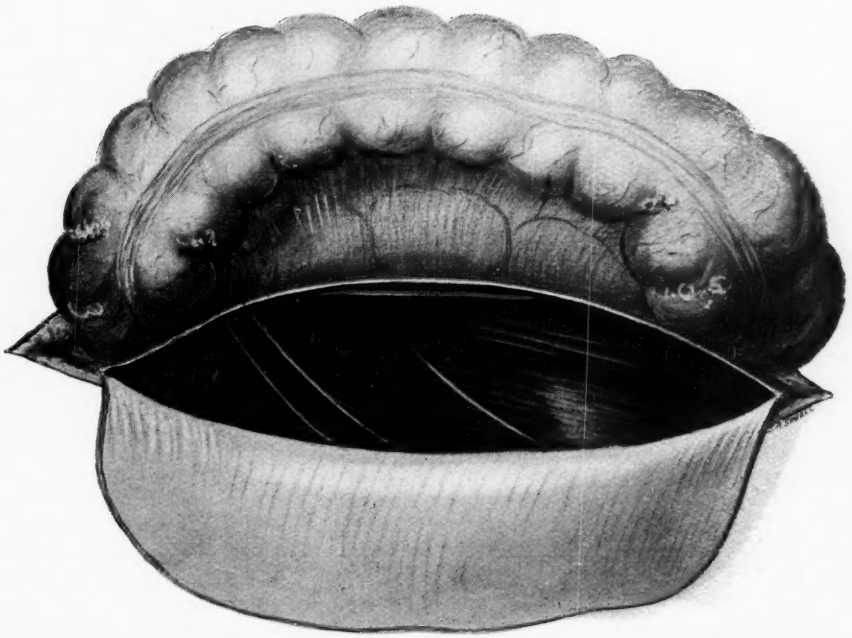


FIG. 360.—The flap of fascia has been raised and turned outwards. The lower pole of the kidney is just seen at the upper end of the bed formed for the colon, the ureter can just be seen mesially, and branches of the lumbar plexus of nerves are visible emerging between the psoas and quadratus lumborum muscles.

(*Fig.* 359). The upper limit of the incision corresponds approximately to the level where the colon crosses the duodenum. Here the fascia becomes much thinned, where it splits to enclose the right kidney. This curtain of peritoneum and fascia is then pulled outwards, and the bed formed by the iliopsoas and quadratus lumborum muscles freed of fat and cellular tissue until the muscle fibres and nerves of the lumbar plexus are seen (*Fig.* 360). Into this bed the colon is then gently coaxed, the cæcum being placed into position first of all. The curtain is pulled over it, and attached by interrupted stitches of catgut to the anterior longitudinal white band of the colon. This is a convenient landmark which ensures the gut being held in proper position without torsion upon its own axis (*Fig.* 361). When gall-stones are also present—as they were in four cases of this series—they are removed in the usual way, and the gall-bladder drained after the fixation of the colon has been completed.

'Jackson's membrane' was only met with once in this series of cases. In cases of the 'gastric type' with a steep ascent of the transverse colon to the splenic flexure (*Fig.* 344),

the omentum is sometimes fixed transversely to the abdominal wall after Coffey's method. The abdomen is then closed in the usual way.

In the earlier cases there was a troublesome tendency to infection of the fat of the abdominal wall with *B. coli*, resulting in the discharge of a stinking hæmatoma about ten days after the operation. There seems to be a peculiar liability to this complication in all operations in which there is prolonged manipulation of the colon, but complete freedom from it has been achieved by the adoption of the following measures. When only the fat layer remains to be closed, it is packed with a large abdominal swab wrung out in 1–20 carbolic lotion. At the same time gloved hands and instruments are well bathed in the same lotion, and clean sterilized towels arranged around the wound, which is then finally closed. More than 100 consecutive cases have now remained free from any *B. coli* contamination of the fat.

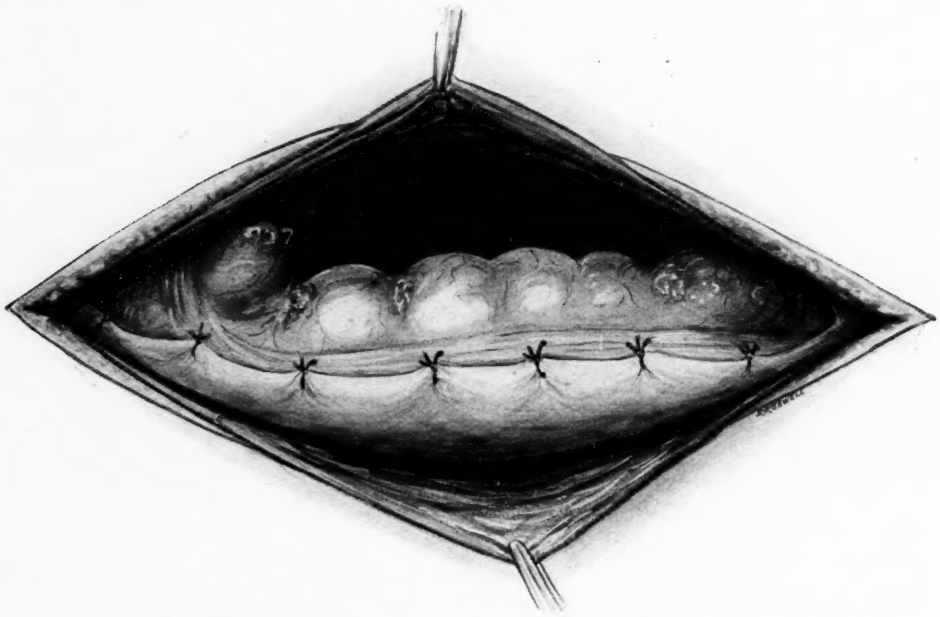


FIG. 361.—The flap of fascia has been fixed to the anterior white line of the ascending colon, except at the hepatic flexure, where the topmost stitch has been fixed to the gut wall above and external to the white line.

After-treatment.—The patient is placed in bed in the Fowler position, and 15 oz. of 20 per cent glucose in saline given by rectum immediately. Half a pint of the same solution is administered every six hours for the next three days. During that time nothing is allowed to be swallowed, especially not sips of water, but the mouth is refreshed by encouraging the patient to chew tinned pine-apple and orange peel, to wash the mouth out with dry ginger ale, eau de Cologne in hot water, and other mouth-washes, and to suck bull's-eye peppermints and acid drops.

An exception to the rule against taking anything by mouth is made in favour of crême-de-menthe, which may be given in teaspoonful doses at intervals. Hypodermic injections of morphia and heroin are used liberally to ensure sleep and freedom from pain. On the fourth day a meal of porridge and cream is given, and three hours later the first cup of tea is allowed. From then onwards the dietary may be increased with rapidity.

No attempt is made to get the bowels to act; they will do so of their own accord when they contain sufficient débris, and when healing of the traumatized peritoneal area has so far advanced that the normal peristaltic reflex is permitted. It is very rare to have troublesome post-operative flatus when this mode of after-treatment is adopted, and should it occur, the use of 4 oz. of a mixture of equal parts of glycerin and water as a high enema, with a simultaneous hypodermic injection of 1 c.c. pituitrin, affords instant relief.

During convalescence, a habit of dry meals, with plenty of fluid in the intervals, is begun, although quite a surprising number of patients had discovered for themselves that this was the most comfortable method of feeding before coming under observation. Special corsets or belts are fitted before the patient is allowed to get up, and instruction given in abdominal exercises for the muscles, to be undertaken two months after operation, and persevered with for six months. Massage and electrical treatment are useful adjuncts for the same purpose when they can be obtained.

RESULTS OF OPERATION.

From an analysis of the types of cases operated upon in adults, the results may be classified as follows: gastric types, 97; duodenal types, 40; biliary types, 11; renal types, 5; right iliac fossa types, 15; gastric ulcers, 4; duodenal ulcers, 6; colitis, 1. Twenty-eight cases are excluded from this list. The operations were performed on them in military hospitals, and, beyond the fact that all of them left the hospitals temporarily relieved of their symptoms, no further record of them can be traced. Including them in the list for reviewing the operative mortality, a total of 208 cases were operated upon, with two deaths as the result of the operation. Of these, one was discovered at the operation to be suffering from an advanced stage of atrophic cirrhosis of the liver (*Case* 73); the other developed general paralytic ileus and died five days later. One case developed a bad ventral hernia, for which a radical cure was subsequently performed. This afforded an opportunity of noting that the ascending colon was firmly fixed in the position in which it had been placed seven months previously. This was also noted in another patient (*Case* 97), upon whom a second laparotomy was done eighteen months later for the removal of breaking-down mesenteric glands.

From the 180 cases here recorded, the appendix had already been previously removed in 68; in 11 the gall-bladder had been drained as well, although gall-stones were only present in one, and the patients had been informed that it was a matter of expediency only, as there was no disease; in a few of them a gastro-enterostomy had also been done; in a few of them the right kidney had been fixed unsuccessfully, and secondary operations for kinks and adhesions performed. In all of these cases a complete fixation of the ascending colon was carried out; an ileal kink was present as well and divided in 9 of them; from 4 of them gall-stones were removed and the gall-bladder drained, although no history of biliary crises was obtainable and the discovery at operation was as unexpected as their formation had been silent; the appendix was removed when present from 112; in 9 cases disease of the appendix was present. In those in whom the appendix is classified as healthy it was small and infantile in appearance, so much so that it almost appeared to be a constant type associated with this class of case, and even the most sceptical would probably have agreed that its mere removal could not be expected to modify any of the patient's symptoms whatsoever; and to be content with such a removal would have been merely to add to the list of patients already grievously disappointed by the failure to obtain relief from their sufferings by appendicectomy.

The results have been recorded as far as possible in the patients' own words. A very large number have obtained a freedom from their previous symptoms. Some, in addition, have markedly improved their general nutrition, as shown by their altered appearance, their augmented capacity for physical endurance, and their increased weight. The duration of the previous illness, and the degree of tissue degeneration already reached, were clearly factors that limited the amount of improvement that might be expected. In only a few of the younger cases with a comparatively short history of illness have the results been poor. That it should happen at all is disappointing, and the reason for the failure has not been obvious.

CONCLUSION.

By whatever stages the upright position may have been attained in man, its final achievement exposed the alimentary tract, no less than the cardiovascular system, to the

maximum effects of the action of gravity. In the quadrupedal position nutriment travels along a horizontal plane with gentle undulations, and save for purposes of absorption which are best served by a convoluted tube, a straight tube from mouth to anus would have no serious mechanical defects. But the biped must not present too steep a descent, with its inevitable acceleration, to the contents of the alimentary tract, nor too abrupt an ascent, with its inevitable retardation of the expulsion of waste material. By a series of curves the human alimentary tract—whether by design or not is immaterial—has achieved a happy compromise between the extremes of acceleration and retardation of its contents, and viewed purely as a problem of mechanics it must be regarded as a mechanism of great efficiency. None the less a flaw in the mechanism, under the inexorable influence of gravity, must sooner or later reveal the individual adjustment to be but an imperfect contrivance. Such a flaw in the vasomotor control of the circulatory system which renders it mechanically efficient against the action of gravity is not compatible with the prolonged survival of the individual in the upright position. A flaw in the mechanical efficiency of the alimentary tract is not compatible with the preservation of health. It may be stated dogmatically that an ascending colon which preserves its primitive mesentery is not a mechanism so mechanically efficient for its task as an ascending colon which has become securely fixed through the disappearance of its mesentery. The toll that is exacted as the penalty of this mechanical inefficiency appears to be represented by the diseases that form the subject of this article.

Now these diseases have two quite distinct, yet intimately related, aspects: the local changes in the viscera that have already been dealt with in detail, and the remoter general changes throughout the tissues of the body. The former appear to be immediately mechanical in origin; the latter to be the results of impaired metabolism, which are the inevitable sequence of mechanical inefficiency. Nothing can be more pitiful than the profound degree of tissue degradation which all these patients present: their discoloured inelastic skins, their emaciation, their irritable hearts, their unstable nervous systems, their enfeebled and easily exhausted musculature. Further, it must be emphasized that most of them had undergone several years of treatment, of most varied and ingenious kinds, directed to one or other of the systems secondarily involved. A modest schedule would include drugs, diet, climate, rest cures, massage, electrotherapy, psychotherapy, colonic lavage, vaccines, good advice, exercises, and hard work.

Toxic End-Products.—It is difficult to avoid the conclusion that these devastating changes in the fabric of the human organism are brought about by the absorption of deleterious end-products from an alimentary tract which is mechanically inefficient for its task, and which suffers, alike with the rest of the body, from its own inefficiency. Hence the failure of these modes of treatment to effect any permanent improvement, since in each case the gross mechanical defect of the alimentary tract was demonstrably present still, in spite of the prolonged course of treatment already experienced.

The formation of toxic end-products is an immediate sequel to the delay in the passage of the contents of the alimentary tract, which in itself depends upon the mechanical inefficiency of the tract to expel them. This delay, as has already been stated, has been noted as an associated condition in all these cases, and it has been explained as a secondary condition arising from the prime mechanical defect; but its importance is not in the least minimized by supposing its origin to have been in this manner.

Intestinal Stasis.—To Arbuthnot Lane we owe the conception of 'intestinal stasis' and the grave consequences that may flow from it. His co-workers have elaborated in detail the chemical nature of the toxic end-products, and their injuriousness cannot be gainsaid. But whilst he has given a masterly conception of the eroding power of 'static' end-products upon the human fabric, the details of his pathology, whereby that stasis has been brought about, have failed to bring conviction, and do not appear to link the train of events together in a logical sequence. 'Kinks,' 'angulation,' and 'constipation' as prime causal agents do not appear sufficiently secure foundations upon which to base an inevitable cycle of events; they are erratic and occasional in their presence, or they may make their appearance only at a late stage of the disease. Nor does the substitution

of a grossly pathological condition, such as is entailed by colectomy or ileocolostomy, make any appeal as a form of treatment likely to be a happy substitute for the ills already endured. With such profound disagreement from the details of his pathology, tribute must nevertheless be paid to the great conception of his views, that the tissues of the human body are most formidably assailed by the toxic end-products of the alimentary tract. To sweep aside these views with petulance will allay neither the intellectual dissatisfaction of the critics nor the physical sufferings of the patients.

It may be a bold claim that the writer is making in the present article that a more lucid conception of the working of 'stasis' has been achieved; that a surgical procedure which is physiological rather than pathological in type is the cure for the ills that arise from it; that an ascending colon which is mobile from birth as a slight congenital defect causes inevitably the train of evils already enumerated; and that the fixation of that part of the colon is to effect a procedure which must prevent these. Yet the improvement which has followed in so many of the cases submitted to operation has given the greatest encouragement to the continuation of work along these lines, and the measure of the success attained is the measure of the debt owing to the original conceptions and views of Arbuthnot Lane.

Ideally, the mobility of the colon should be recognized in childhood and the operation performed then. It is a much smaller procedure in children, as a pseudo-mesentery has not yet made its appearance, and the operation is well tolerated by them. No death has occurred in nearly 100 cases of children submitted to operation. If performed during this period of life there is not only a reasonable expectation of a better degree of health during childhood, but also the hope that the digestive disorders of adolescence and the end-stage diseases of the adult may be entirely prevented. The results show that the earlier in life the operation was performed, the greater the success that attended it.

Several cases were submitted to operation after life-long suffering, and in a condition of profound tissue degeneration, on their own earnest entreaty. Many of them got some benefit, but it is clearly most undesirable that they should be regarded as the type for operation after thirty years' trial of other remedies. There is a stage in every case in which tissue degeneration has reached a point beyond all hope of recovery, and it is only previous to that stage that surgical success may be expected.

Reluctance to undergo surgical treatment may be quite reasonably entertained if it involves the removal of a portion of the body, or a gross alteration of the normal relationship of the viscera, and there is a very natural inclination to postpone such an experience until its avoidance is no longer possible. Fixation of the ascending colon is attended by neither of these disabilities, and the strongest plea to be urged on behalf of the operation is that the individual is nearer to the normal type after its performance than he ever was before. It is an operation which is essentially physiological and constructive, and its purpose is to enable the individual to maintain the functions of the alimentary tract unimpaired by the action of gravity to which the biped is exposed.

The defective condition can be diagnosed with ease and certainty in every patient examined, and when its presence has been clearly proven, an early surgical treatment is essential. In the adolescent, with a comparatively short history of illness, the results of the operation have been most encouraging, whilst its effectiveness has been clearly diminished in proportion to the length of time of the previous illness and symptomatic treatment. No attempt has been made in estimating the results to draw up percentage lists of successes and failures. The patients have been written to, and their own views in their own words about the consequences of the operation have been recorded in the end column of the table of cases under the heading of 'Result.'

To many doctors I am deeply indebted for their collaboration in obtaining this information, and my special thanks are due to Drs. H. Huxley, W. McD. Ellis, W. Jagger, J. T. C. Laing, A. Gale, and D. Pennington. The sketches have been made from actual operations by Mr. Sydney Sewell, who has devoted much time and skill to their production.

[*Record of Operations on next page.*

RECORD OF 180 OPERATIONS FOR MOBILE ASCENDING COLON.

No.	Case	Sex	Age	Type	Duration of Previous Illness and Treatment	Operation	Result
						1911	
* 26	A.H.	F.	45	Duodenal	Many years hunger pains with every variety of treatment	Simple appendicectomy. No ulcer. Colopexy. Nov. 10	Reported 'very well 3 years later'
						1912	
27	R.B.	F.	35	Gastric	3½ years epigastric pain. Treated by diet and drugs	Healthy appendix removed. Ascending colon fixed. Feb. 2	Quite well 2 years later
28	A.T.	F.	31	Gastric	3½ years pain. 2 attacks of colitis	Healthy appendix removed. Colopexy. March 4	'No further trouble inside.' Reported June, 1916
29	K.H.S.	M.	43	Biliary	10 years gross dyspepsia. Appendicectomy 5 years ago. Acute biliary crisis on May 14 lasting 48 hours	Colopexy. No gall-stones. May 24	Report April, 1919. No attack since operation. Dyspepsia much better, but not quite cured
30	R.T.	M.	20	R. iliac fossa	1 year nausea, with pain in right iliac fossa, attacks lasting	Small appendix removed. Ileal kink freed. Colopexy. June 20	Seen Aug. 17, 1915. 'Quite well'
31	A.B.	F.	31	Gastric	Complete invalid for 7 years. Appendicectomy 5 years ago. Lives on ground floor, unable to walk upstairs. Several attacks of colitis	Colopexy. May 7	Report July, 1918. 'Was riding on horseback and quite well. No further attacks of colitis'
						1913	
32	S.T.	F.	27	Gastric	5 years gastric pain. Appendicectomy 4 years ago. No relief	Colopexy. Nov. 15	Report Nov., 1917. 'Quite well'
33	R.L.	F.	44	Gastric	Under treatment since 1902. Appendicectomy 1903. Condition gradually getting worse	Colopexy. Nov. 15	Report July, 1917, 'General health much improved, leads an active life'
						1914	
34	R.W.	M.	34	Biliary and Gastric	5 years dyspepsia. Appendicectomy 3 years ago. History of acute crises, biliary crisis seen on Feb. 5, 1914	Colopexy. No gall-stones. Feb. 23	Report May, 1918, 'In perfect health and has put on 2 stones in weight'
35	S.H.	F.	29	R. iliac fossa	14 months chronic pain right iliac fossa. Nausea lasting for days, has lost 20 lb. in weight during this time, occasional diarrhœa	Small healthy appendix removed. Colopexy. March 2	Report June, 1917, 'No further attacks since operation and has put on 1 stone in weight'
36	M.S.	F.	30	Gastric	Dyspepsia several years, pain 6 months, many attacks of colitis, 1 year invalidism	Simple healthy appendix removed. Colopexy. June 11	Writes, Oct., 1919, 'Health quite normal and leading an active life'
37	F.D.	M.	32	Gastric	3 years symptoms. Many forms of treatment	Apparently healthy appendix removed, ileal kink undone. Colopexy. July 28	Got quite well. Was killed in action Sept., 1917
						1915	
38	J.M.	M.	21	Duodenal	18 months pain and indigestion	Healthy appendix removed. Ileal	Returned to active service 10

					of treatment	moved, ileal kink undone. Colopexy. July 28	action Sept., 1917
						1915	
38	J.M.	M.	21	Duodenal	18 months pain and indigestion	Healthy appendix removed. Ileal kink cut. Colopexy. Jan. 11	Returned to active service 10 months later. Killed, July, 1916
39	W.W.W.	M.	42	Duodenal	10 years hunger pains and in right iliac fossa. Appendicectomy 8 years ago. No improvement	Colopexy. Jan. 28	Report June, 1917. Very much better. Very occasional dyspepsia, but quite free from pain.
40	A.G.Q.	M.	23	R. iliac fossa	2 years dyspepsia. Nausea lasting several days	Healthy appendix removed. Colopexy. March 6	Went on active service in Nov., 1915, and reported 1 year later, 'In excellent health'
41	A.R.	M.	27	R. iliac fossa	Appendicectomy for chronic pains 3 years ago. No relief	Colopexy. March 8	Returned to active service at front 6 months later. No further report
42	S.L.	M.	25	Duodenal	4 years dyspepsia and hunger pains	Healthy appendix removed. Colopexy. April 12	Left hospital well, but not traced afterwards
43	A.S.	F.	29	Gastric	'Always had dyspepsia since about 20 years of age.' Appendicectomy a few years ago. No relief	Colopexy. Nov. 13	Report 2 years later. 'Very well and doing nursing abroad'
44	M.F.	F.	27	Duodenal	Several years dyspepsia. Pain 2 years, Appendicectomy 4 years ago	Colopexy. Nov. 13	Report 1 year later. 'Quite well'
						1916	
45	R.P.	M.	25	Duodenal	1 year dyspepsia and hunger pains	Healthy appendix removed. Colopexy. Feb. 22	Left hospital quite well. Killed in France, July, 1917
46	S.R.	M.	37	Duodenal	Dyspepsia some years. Hunger pains 2 years. Appendicectomy 2 years ago	Colopexy. March 6	Was quite well 6 months later. Not heard of since.
47	S.H.	M.	37	Duodenal	Gastro-enterostomy and appendicectomy 8 years ago. No relief	Colopexy, no ulcer found. March 13	Put on 1 stone in weight in following 8 months. Free from symptoms. Not heard of afterwards
48	J.McK.	M.	32	Gastric	5 years dyspepsia and gastric pain. Appendicectomy 6 years ago	Colopexy. March 25	Reported 'very well' six weeks later. Not traced since
49	R.L.	M.	28	R. iliac fossa	Chronic pain right side 2 years. Appendicectomy 6 months previously	Colopexy. June 12	Put on 1 stone in weight in a few months, but said that he was 'no better' and obviously intended to get out of the Army
50	L.H.	M.	34	Gastric	Dyspepsia many years. Appendicectomy 7 years ago	Colopexy. June 19	Known to be well for 1 year
51	M.J.	M.	27	Duodenal	2 years pains and dyspepsia. Appendicectomy 18 months ago. No relief	Colopexy. June 29	Very well 8 months later, but not traced since
52	R.A.	M.	35	Gastric	'Dyspepsia' many years. Appendicectomy 14 years ago	Colopexy. July 17	Reported well 10 months later and went abroad
53	A.M.	F.	30	Gastric	Always had dyspepsia. Pain several years	Healthy looking appendix removed. Colopexy. July 31	Seemed very well while in hospital, but not traced since

* Nos. 1 to 25 are detailed in the text.

Continued on next page

RECORD OF 180 OPERATIONS FOR MOBILE ASCENDING COLON—*continued.*

No.	Case	Sex	Age	Type	Duration of Previous Illness and Treatment	Operation	Result
54	A.M.	F.	18	Gastric	Always delicate since a child. Appendix removed 11 years old. No improvement	Colopexy. Aug. 2	Report July, 1918, 'Quite well and had put on 1 stone in weight'
55	T.J.	M.	28	Duodenal	4 years dyspepsia. 2 years pain. Appendicectomy 2 years ago. No improvement	Colopexy. Aug. 3	Very well at end of two months. Not traced after leaving hospital
56	T.L.	M.	26	Duodenal	2 years symptoms	Appendix with adhesions around it removed. Colopexy. Aug. 14	Quite well, and a few months later returned to active service
57	F.S.	M.	27	Gastric	'Always trouble with inside.' Appendicectomy 7 years ago	Colopexy. Aug. 18	Well and returned to active service
58	N.H.	M.	29	Gastric	Some years of dyspepsia	Healthy appendix removed. Colopexy. Aug. 18	Well and returned to active service
59	R.N.	M.	26	R. iliac fossa	Chronic pain and dyspepsia 6 years. Always tired. Nausea	Appendix with adhesions removed; ileal kink cut. Colopexy. Aug. 21	Very well at end of 7 months. Not traced since
60	J.D.	F.	26	Gastric	Dyspepsia 5 years. Appendicectomy 4 years ago. No relief	Colopexy. Aug. 23	Quite well to when seen 1 year later, and had put on weight
61	G.N.	M.	25	R. iliac fossa	3 years indigestion and discomfort in right iliac fossa	Healthy appendix removed. Colopexy. Sept. 11	'Well and returned to active service Oct., 1919, quite well'
62	T.L.	M.	26	Duodenal	Some years suffering. Appendicectomy when 8 years old. No relief	Colopexy. Oct. 5	Left hospital very well at end of 3 months
63	R.S.	M.	29	Duodenal	3 years dyspepsia and hunger pains	Healthy appendix removed. Colopexy. Oct. 9	Left hospital very well, but not traced after 4 months
64	H.T.	F.	18	R. iliac fossa	Pain 2 to 3 years' duration, lasts several days, and relieved by diarrhœa	Embedded unhealthy appendix removed. Colopexy. Oct. 11	Reported May, 1919, 'Quite well'
65	R.T.	M.	29	Gastric	Several years dyspepsia. Gastric pain 3 years	Healthy appendix removed. Colopexy. Oct. 13	Writes, Nov. 19, 1919, 'Quite fit. I have been wounded on 6 different occasions since the operation.'
66	H.W.	M.	28	Duodenal	3 years dyspepsia and hunger pains	Healthy looking appendix removed. Colopexy. Oct. 30	Well at end of 2 months. Not traced after leaving hospital
67	F.W.	F.	24	Gastric	Chronic gastric pain and dyspepsia for several years	Unhealthy appendix removed and ileal kink cut. Colopexy. Dec. 29	June, 1918, 'Improved, but digestion still very troublesome at times'
68	A.S.	F.	32	Gastric	Chronic pains and dyspepsia for many years, much loss of weight and lassitude. Appendix removed 4 years previously	Colopexy. Dec. 29	Seen Feb., 1919, 'Very well, has put on weight and can lead an active life free from pain'
69	C.N.W.	F.	28	Gastric	Many years dyspepsia. Treated for sharp gastric pain for about 3	Simple appendix removed. Colopexy. Dec. 29	Nov., 1919. 'Leads an active life. Has some dyspepsia still, but

					many years, much loss of weight and lassitude. Appendix removed 4 years previously	Colopexy. Dec. 29	Seen Feb., 1919, 'Very well, has put on weight and can lead an active life free from pain'
69	C.N.W.	F.	28	Gastric	Many years dyspepsia. Treated for sharp gastric pain for about 3 years	Simple appendix removed. Colopexy. Dec. 29	Nov., 1919. 'Leads an active life. Has some dyspepsia still, but quite free from gastric pain'
						1917	
70	L.W.	M.	23	Gastric	Dyspepsia and pain 1 year. Discomfort in right iliac fossa always present	Healthy appendix removed. Colopexy. Jan. 19	Quite well 1 year later
71	R.H.	M.	27	Gastric	Dyspepsia and pain on and off for a few years. Loss of weight more than 1 stone	Appendix with adhesions removed, Colopexy. Jan. 22	Writes, Oct., 1919, 'Better than I have ever been'
72	T.C.	M.	27	Duodenal	Dyspepsia for 5 years, hunger pains 1½ years	Healthy appendix removed. Colopexy. March 28	Reported 5 months later, very well and on active service
73	R.C.	M.	49	Gastric	Many years dyspepsia, chronic pain right iliac fossa and right hypochondrium	Healthy appendix removed. Liver found to be small and shrunken, clearly advanced atrophic cirrhosis. Colopexy. April, 1916	Died 1 week after operation with uræmic convulsions
74	J.D.	M.	24	R. iliac fossa	Dyspepsia some years. Chronic pain 1 year	Healthy appendix removed. Ileal kink cut. Colopexy. April 16	Dec., 1918, reported 'Quite well'. Dec., 1919, 'Nerves rather bad'
75	B.R.	F.	31	Gastric	Dyspepsia 3 years, pain about 2 months	Healthy appendix removed. Colopexy. April 17	Writes, Oct., 1919, 'Perfectly well. I have no further trouble of any sort'
76	T.C.	M.	25	Duodenal	Dyspepsia 3½ years, hunger pains 1½ years. Appendicectomy	Colopexy. April 19	Writes, Oct., 1919, 'Present condition excellent'
77	M.D.	M.	27	Gastric	Had always suffered from stomach trouble, which became very acute Jan., 1917. Appendix removed 6 years ago	Colopexy. May 11	Writes, Oct., 1919, 'Digestion better than it has ever been in my life before'
78	R.B.	M.	22	Gastric	Dyspepsia 3 years	Healthy appendix removed. Ileal kink cut. Colopexy. June 13	Very well 6 months later
79	D.P.	F.	21	Gastric	Dyspepsia and attacks of pain for 3 years	Simple appendix removed. Colopexy. June 15	Oct., 1919, writes, 'Very fit; have had splendid health since the operation'
80	T.W.	M.	25	R. iliac fossa	Chronic pain several years. 'Periods of nausea lasting for several days'	Healthy appendix removed. Colopexy. June 25	Seen in Jan., 1918. 'Said he was much better and had no pain, but occasional gastric flatulence'
81	T.F.	M.	27	R. iliac fossa	Dyspepsia and chronic discomfort right iliac fossa, 2 years	Healthy appendix removed. Colopexy. July 7	Reported 2 years later, 'Some improvement, but digestion not very good'
82	L.B.	M.	23	Duodenal	Hunger pains and dyspepsia 1 year	Healthy appendix removed. Colopexy. July 27	Very well 5 months later, and returned to active service
83	V.DeS.	M.	31	Duodenal	Dyspepsia some years, hunger pains 3 years	Healthy appendix removed. Colopexy. Sept. 2	March, 1918. Reported 'Quite well'
84	F.R.	M.	33	Gastric	Dyspepsia many years. 3 attacks of colitis	Healthy appendix removed. Colopexy. Sept. 7	Sept. 13. Died of general paralytic ileus

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RECORD OF 180 OPERATIONS FOR MOBILE ASCENDING COLON—*continued.*

No.	Case	Sex	Age	Type	Duration of Previous Illness and Treatment	Operation	Result
85	T.B.	F.	36	Gastric	Dyspepsia and constantly recurring attacks of colitis 6 years' duration. Appendix removed 4 years ago	Colopexy. Oct. 13	Writes, Oct., 1919, 'Attacks of colitis have ceased, but digestion not entirely satisfactory'
86	H.C.G.	M.	41	Duodenal	18 months hunger pain. Dyspepsia several years	Healthy appendix removed. Colopexy. Oct. 28	Writes, Oct. 1, 1919, 'I am very well, and digestive organs give no trouble'
87	E.G.	F.	31	Gastric and R. iliac fossa	Chronic ill health for 10 years, pain 2 years. Appendix removed 5 years ago; no improvement	Colopexy. Nov. 8	Writes, Oct., 1919, 'General health enormously improved'
88	J.P.	F.	19	Gastric	Dyspepsia 3 years. Pain 1 year. Much lassitude	Healthy appendix removed, ileal kink cut. Colopexy. Nov 29	'Much improved' 1 year later
89	L.R.	F.	35	R. iliac fossa	Dyspepsia and chronic pain 6 months. Prolonged attacks of nausea	Healthy appendix removed. Colopexy. April 30	Writes, Nov., 1919, 'I have nothing wrong with me at all'
90	E.K.	F.	31	Gastric	Many years dyspepsia and pain. Appendicectomy 3 years ago. Has lived as an invalid for last year entirely	Colopexy. April 11	Writes, Nov., 1919, 'I do not seem to be any better for the operation'
91	H.B.	F.	29	Duodenal	7 years treatment for indigestion and hunger pains. Appendicectomy 6 years ago	Colopexy. Oct. 17	Writes, Dec., 1919, 'Very well indeed'
92	J.S.	F.	36	Gastric	Many years suffering. Right kidney fixed 1907, Appendicectomy 1913. Colitis several times	Colopexy. Dec. 7	Dec., 1919, 'I still have indigestion but I am much stronger'
93	A.F.	F.	35	Duodenal	2 years pain. Dyspepsia many years	Healthy appendix removed. Colopexy. Dec. 14	Dec., 1919, writes, 'No pain and much stronger in every way'
						1918	
94	E.H.	F.	32	Gastric	Dyspepsia some years. Pain 2 years, 2 attacks colitis. Enlarged thyroid several months	Healthy appendix removed. Colopexy. Jan. 11	Writes, Oct., 1919, 'Better than I have been for years'
95	H.F.	F.	37	Gastric	Dyspepsia some years. Removal of appendix gave no relief. Gastric pain 6 months	Colopexy. Jan. 2	Writes, Oct., 1919, 'Perfectly all right, no pain anywhere'
96	L.H.	M.	27	Gastric	5 years indigestion and pain. Appendicectomy 5 years ago. No relief	Colopexy. Jan. 4	May, 1919. Very well
97	D.S.	F.	19	Gastric	Invalid since childhood. Many attacks of pain in right hypochondrium and right iliac fossa. Always under treatment	Healthy appendix removed. Colopexy. Jan 4	Very well for 9 months. Nursed as V.A.D. 6 months, then different pains returned. Laparotomy July, 1919. Large ovarian cyst removed, and breaking down glands from mesentery. Ascending

					attacks of pain in right hypochondrium and right iliac fossa. Always under treatment	pexy. Jan 4	V.A.D. 6 months, then different pains returned. Laparotomy July, 1919. Large ovarian cyst removed, and breaking down glands from mesentery. Ascending colon seen to be firmly fixed in position. Dec., 1919, 'Leading an active life, quite free from pain
98	H.H.	F.	39	Gastric	Always delicate. Pain much worse last 18 months	Simple appendix removed. Colopexy. Jan. 5	Writes, Oct., 1919, 'Very much improved, and am able to bicycle many miles without fatigue'
99	M.M.	F.	39	Gastric	Always suffered from chronic pains and dyspepsia. Appendicectomy some years ago. Very enervated	Colopexy. Jan. 11	July, 1919. No better, has had transient hemiplegia
100	G.M.	M.	31	Gastric	4 years dyspepsia. Pain 1 year	Diseased appendix removed. Colopexy. Jan. 14	Writes, Oct., 1919, 'No better at all, but think that any troubles may arise from shell shock'
101	C.S.	M.	29	Duodenal	3 years dyspepsia, pain about 18 months. Appendicectomy 5 years ago	Colopexy. Jan. 16	Feb., 1919, reported 'Quite well'
102	H.F.	M.	22	Duodenal	Dyspepsia and hunger pains 1 year. Appendicectomy 3 years ago	Colopexy. Jan. 17	June, 1919. Report, 'Very well'
103	G.S.	F.	35	Gastric	Has suffered since childhood, and during previous year had much severe pain	Small healthy appendix removed. Colopexy. Jan. 18	Writes, Oct., 1919, 'Condition very excellent, no pain, can eat anything, and am 1 stone heavier than I have ever been'
104	H.L.	F.	44	Gastric	Dyspepsia on and off 15 years, much worse lately. Pain 5 months. Colitis 1 year	Healthy appendix removed. Colopexy. Feb. 27	Writes, Nov., 1919, 'Much better; much less gastric pain; no diarrhœa, but inclined to constipation
105	H.H.	F.	34	Gastric	8 years chronic pain and indigestion. Appendix removed 5 years ago	Colopexy. Feb. 15	Writes, Oct., 1919, 'General health better than it has been for years'
106	G.E.	F.	21	Gastric and R. iliac fossa	18 months dyspepsia, with attacks of gastric pain, followed by extreme discomfort in right iliac fossa	Simple appendix removed. Colopexy. Feb. 22	Writes, Oct., 1919, 'Quite fit: much better since operation'
107	L.S.	F.	34	Gastric	Has never been free from abdominal pain and discomfort since 14 years of age. Many attacks of colitis. Appendicectomy 4 years ago; no improvement	Colopexy. Feb. 28	Writes, Oct., 1919, 'Very greatly improved. I am in better health than I have been for many years'
108	J.M.	M.	51	Gastric	Has always had dyspepsia. Pain about 1 year	Healthy appendix removed. Colopexy. March 1	Writes, Oct., 1919, 'Much improved in bodily endurance and freedom from headaches'
109	A.A.	F.	40	Duodenal	Many years pain and dyspepsia. Appendicectomy and drainage of gall-bladder after removal of stones 6 years ago. Relief for 3 weeks only	Colopexy. March 5	Writes, Oct. 1, 'Much stronger than ever before'

Continued on next page

RECORD OF 180 OPERATIONS FOR MOBILE ASCENDING COLON—*continued.*

No.	Case	Sex	Age	Type	Duration of Previous Illness and Treatment	Operation	Result
110	A.W.	F.	21	Gastric	2 years bad indigestion and pain. Continuous treatment. Has lost 2 stones in weight	Healthy appendix removed. Colopexy. March 9	Writes, Oct. 1, 'General health much improved, and free from indigestion. Dec., 'Pain has come back again'
111	G.H.	M.	39	Duodenal	Many years dyspepsia and pain. Occasional colitis. Attacks of tachycardia	Healthy appendix. Colopexy. March 11	Died 6 months later of an intercurrent illness. No pain since operation
112	J.B.	M.	17	Gastric	2 years dyspepsia, with occasional attacks of pain. Losing weight	Healthy appendix removed. Colopexy. March 25	June, 1919, 'Very well'
113	G.M.	M.	32	Duodenal	4 years dyspepsia. Pain 2½ years. Attacks of diarrhœa	Healthy appendix removed. Colopexy. April 16	Writes, Oct. 1, 'Good so far as digestion is concerned, no pain, and physical condition much better'
114	E.H.	F.	41	Duodenal	Dyspepsia and hunger pains several years. 4 attacks of colitis in last 2 years. Appendicectomy and drainage of gall-bladder 7 years ago	Colopexy. May 11	April, 1919. 'Still has some flatulence, but is better than she ever has been'
115	Q.R.	F.	24	R. iliac fossa	2 years chronic discomfort. Attacks of nausea	Congested appendix with adhesions removed. Colopexy. May 21	Writes, Oct., 1919, 'Quite fit'
116	E.H.	F.	41	Gastric	Has always suffered from indigestion. Pain last 5 years	Healthy appendix removed. Colopexy. May 21	Seen Oct. 16, 1919. 'No better at all since the operation, and her general condition is very unsatisfactory'
117	J.P.	F.	31	Gastric	15 years dyspepsia and treatment. Appendicectomy 9 years ago	Colopexy. May 30	Aug., 1919, writes, 'Very much better in every way, and only occasional flatulence'
118	C.M.	F.	30	Gastric	Pain 4 years duration. Dyspepsia some years previously	Appendix removed, ileal kink cut. Colopexy. June 13	Remained quite well till March, 1919, when she had an attack of jaundice. Seen in July, 1919, and says she is much better again
119	H.J.	F.	29	Gastric	Pains and dyspepsia ever since a schoolgirl. Always ill and under treatment	Healthy appendix removed. Colopexy. July 4	Writes, Oct., 1919, 'General condition enormously improved'
120	M.P.	F.	33	Gastric	Treated for gastric ulcer for 3 years—5 acute attacks. Dyspepsia in intervals	Healthy appendix removed. No gastric ulcer. Colopexy. Aug. 10	Writes, Oct., 1919, 'General condition much improved, but still subject to dyspepsia and constipation'
121	F.G.	M.	29	Gastric	Indigestion since 1912, bad pain last 6 months. Has been under treatment for gastric ulcer	Appendix removed. No ulcer. Colopexy. Aug. 30	Remained quite well 3 months under observation. No trace subsequently
122	M.W.	M.	41	Gastric	Under treatment 15 years. 'Steadily getting worse' (medical	Simple healthy appendix removed. Colopexy. Sept. 10	April, 1919, 'Has never been so well in his life, and considers his

121	F.G.	M.	29	Gastric	Indigestion since 1912, bad pain last 6 months. Has been under treatment for gastric ulcer	Appendix removed. No ulcer. Colopexy. Aug. 30	Remained quite well 3 months under observation. No trace subsequently
122	M.W.	M.	41	Gastric	Under treatment 15 years. 'Steadily getting worse' (medical practitioner)	Simple healthy appendix removed. Colopexy. Sept. 10	April, 1919, 'Has never been so well in his life, and considers his digestion is superior to that of the average individual'
123	M.B.	F.	48	Gastric	Many years pain and dyspepsia, 3 attacks colitis, much emaciation	Healthy appendix removed. Colopexy. Sept. 18	Oct., 1919, 'Still much trouble with dyspepsia and flatulence'
124	C.F.	F.	42	Gastric	Many years of continuous suffering—profound emaciation, and living on slops last year, persistent colitis during past year.	Healthy appendix removed. Colopexy. Oct. 3	Oct. 7, 1919, 'Has had only 1 attack of colitis since operation; feels much better, can enjoy her food, and walk further than she was able to do; still has some dyspepsia and flatulence at intervals, but no pain'
125	E.S.	F.	52	Gastric	Chronic invalid all her life	Healthy appendix removed. Colopexy. Oct. 16	Oct., 1919, 'Still weak, and a good deal of trouble with indigestion'
126	A.M.	F.	42	Gastric	23 years dyspepsia and pain. Appendicectomy and gall-bladder drainage 6 years ago; no calculi	Colopexy. Oct. 30	Writes, Oct. 2, 1919, 'Better and fatter since operation. General tone improved'
127	P.D.	F.	37	Gastric	Several years dyspepsia. Attacks of severe biliary pains 3 times. Appendicectomy some years ago	Colopexy. Oct. 26	Aug. 1919, seen and found to be very well
128	E.T.	F.	45	Duodenal	25 years chronic suffering, pain, dyspepsia, 1 attack of renal colic. Appendicectomy and gall-bladder drainage some years ago	Colopexy. Jan. 5, 1919	Writes, Dec., 1919, 'I have no pain, and altogether much better, but still constipated'
129	A.M.	F.	44	Gastric	Under treatment continuously for last 10 years	Simple appendix removed. Colopexy. Nov. 2	Writes, Dec., 1919, 'I am much better and have put on weight'
130	D.B.	F.	40	Gastric	Invalid for 10 years. Lying down for 2 years. Appendicectomy 6 years ago. No relief	Colopexy. Nov. 7	Very well, and leading an active life when seen July, 1919
131	H.R.	F.	40	Duodenal	Treated for duodenal ulcer 2 years. Lost weight, dyspepsia several years previously. No better	Healthy appendix removed. No ulcer. Colopexy. Nov. 22	Writes, Dec., 1919, 'Still dyspepsia, but no pain'
132	D.S.	F.	31	Gastric	Pain and dyspepsia for 11 years, much worse during last 5 years	Simple appendix removed. Colopexy. Nov. 11	Writes, Oct. 1, 1919, 'Very much improved'
133	E.R.	F.	20	Gastric	Dyspepsia 4½ years, gastric pain 6 months	Healthy appendix removed. Colopexy. Nov. 26	Writes, Oct. 1, 1919, 'Very much better in every way'
134	G.A.	M.	37	Duodenal	Many years dyspepsia; has been treated as a duodenal ulcer for last 2½ years; getting worse	Simple appendix removed. No ulcer. Colopexy. Dec. 6	Well for 3 months and then began to suffer again from dyspepsia; seen in July, 1919, with a very large ventral hernia; radical cure July 22, 1919; colon seen to be firmly fixed in position. Writes, Nov. 11, 1919, 'The old symptoms have entirely disappeared. I have had no pain for some months. I am leading an active life'

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RECORD OF 180 OPERATIONS FOR MOBILE ASCENDING COLON—*continued.*

No.	Case	Sex	Age	Type	Duration of Previous Illness and Treatment	Operation	Result
135	A.P.	M.	26	Gastric	16 months dyspepsia and gastric pain, followed by discomfort in right iliac fossa	Diseased appendix removed. Colopexy. Dec. 12	Quite well 4 months later. Not traced since
136	J.W.	M.	28	Gastric	Several years dyspepsia and pain. Innumerable attacks of colitis	Healthy appendix removed. Colopexy. Dec. 15	Nov., 1919, 'No more colitis and feel stronger, but digestion is not perfect'
						1919	
137	A.P.	F.		Gastric	12 years dyspepsia. Appendix removed several years ago. Pain on and off last 5 years	Colopexy. Jan 8	
138	E.T.	F.	45	Gastric	Always trouble with inside, under treatment for 25 years. Much worse last 5 or 6 years	Simple appendix removed. Colopexy. Jan 8	Nov. 27, writes, 'I have no pain now, and altogether much more life in me. Have put on 8 lb. in weight'
139	R.M.	M.	25	Gastric	Dyspepsia 4 years, has had several attacks of gastritis. Appendicectomy 3 years ago	Colopexy. Jan. 11	Very well 3 months later
140	P.D.	F.	41	Gastric	Pain on and off last 3 years, dyspepsia for several years. Colitis at intervals	Simple appendix removed. Colopexy. Jan 15.	Writes, Oct., 1919, 'Very well, no pain and very active'
141	F.P.	M.	26	Gastric	6 years dyspepsia and pain, 1 attack of colitis	Healthy appendix removed. Colopexy. Jan 21	Writes in June, 1919, 'that he is no better at all for the operation'
142	A.T.	F.	27	Gastric	Dyspepsia 10 years, pain 1 year, culminating in complete breakdown, Oct., 1918	Healthy appendix removed. Colopexy. Jan 22	Writes, Oct., 1919, 'Feel much better, inside quite all right now, and gives no trouble. I can take an interest in life again'
143	R.B.	M.	29	Gastric	Dyspepsia for about 5 years: regurgitation of food; does not vomit, but brings up undigested food in quantities during the course of a meal; has been subject to this for 6 months, and is getting much worse	Simple appendix removed. Colopexy, Jan. 24	Seen in June, 1919, when he said he was quite well and had put on 6 lb. in weight. Oct. 1, 1919, writes, 'Improve daily, no regurgitation, and feel quite normal'
144	F.W.	M.	29	Gastric	Always been delicate and much troubled with dyspepsia, very much worse last few years; appendix removed several years ago	Colopexy. Feb. 4	Writes, Oct. 1 1919, 'Not much improvement yet'
145	L.B.	F.	40	Gastric	Many years suffering; has been lying down for last 2 years, leading an invalid's life. Appendix removed 6 years ago, no improvement	Colopexy. Feb. 12	Seen in Aug., 1919. 'Leading an active life, and had put on 8 lb. in weight'
146	M.M.	F.	43	Gastric	Has been under treatment continuously last 10 years: steadily losing ground	Simple appendix removed. Colopexy. Feb. 17	Writes, Dec., 1919, 'I am thankful to say I am much better and able to do much more and have put on

					removed 6 years ago, no improvement		
146	M.M.	F.	43	Gastric	Has been under treatment continuously last 10 years: steadily losing ground	Simple appendix removed. Colopexy. Feb. 17	Writes, Dec., 1919, 'I am thankful to say I am much better and able to do much more and have put on weight'
147	A.R.	F.	22	Duodenal	Many years dyspepsia, hunger pains 18 months; said to have a duodenal ulcer	Simple appendix removed. No ulcer. Colopexy. Feb. 19	Writes, Oct. 1, 1919, 'Feeling ever so much better, and I have been riding'
148	A.S.	F.	26	Gastric	Dyspepsia 5 years. Pain last few months	Simple appendix removed. Colopexy. March 8	Nov., 1919, 'Very comfortable inside'
149	F.L.	F.	42	Duodenal	Always dyspepsia, very much worse last few years, appendix removed several years ago	Colopexy. March 22	Writes, Oct. 1, 'Very much better, occasional slight discomfort, but periods of feeling very well which I never experienced before the operation'
150	A.P.	F.	37	Gastric	Gastric pains 2 years, dyspepsia many years, appendix removed 1917, no relief at all	Colopexy. March 22	Writes, Oct. 15, 'Ever so much better, but still occasional dyspepsia
151	M.S.	F.	24	Gastric	Much pain last 3 years, dyspepsia several years, appendix removed 2 years ago, but no relief	Colopexy. April 2	Writes, Oct. 15, 1919, 'Ever so much better, but still occasional dyspepsia
152	T.T.	M.	27	Gastric	Many years of pain and dyspepsia	Simple appendix removed. Colopexy. April 19	Oct., 1919, 'Very well and good digestion'
153	A.C.	F.	43	Gastric	10 years dyspepsia, gastric pain 18 months; never jaundice on any acute attack	Simple appendix removed, gall-stones found and gall-bladder drained after cutting band between it and hepatic flexure. Complete mesentery to ascending colon. Colopexy. April 30	Dec., 1919, 'Very well so far. Occasional flatulence'
154	M.T.	F.	25	Gastric	4 years pain and dyspepsia, lost 15 lb. in weight. Appendix removed 2 years ago	Colopexy. May 1	Dec., 1919, 'Very well and practically no trouble'
155	R.N.	F.	22	Duodenal	2 years pain	Simple appendix removed. Colopexy, May 10	Dec., 1919, 'No pain and much better'
156	M.H.	F.	24	Gastric	Always delicate	Simple appendix removed. Colopexy. May 12	Dec., 1919, 'Much better'
157	E.L.	F.	25	Gastric	Many years indigestion, pain 2 years, high degree of debility	Simple appendix removed. Colopexy. May 14	Writes, Aug., 1919, 'Better than I have been for years'
158	M.B.	F.		Gastric and R. iliac fossa	2 years dyspepsia and pain in gastric region, lasted about 2 hours and was then suddenly relieved, when discomfort in right iliac fossa became evident	Simple appendix removed. Colopexy. May 21	Seen Dec., 1919. Has had no pain since operation and feels much stronger

Continued on next page

RECORD OF 180 OPERATIONS FOR MOBILE ASCENDING COLON—*continued.*

No.	Case	Sex	Age	Type	Duration of Previous Illness and Treatment	Operation	Result
159	A.G.	F.	25	Gastric	Dyspepsia and pain 7 years; lost 3 stone in weight since 1917	Simple appendix removed. Colopexy. May 29	Dec., 1919, 'Leading an active life'
160	A.P.	F.	37	Duodenal	Several years dyspepsia. Pain 18 months	Simple appendix removed, 1 large gall-stone removed from gall-bladder, which was then drained. Colopexy. June 2	Dec., 1919, 'Very well'
161	W.C.	M.	35	Duodenal	12 years dyspepsia and pain, profound debility, one attack of jaundice with great pain; appendix removed some years ago	Band to healthy gall-bladder cut. Colopexy. June 5.	Dec., 1919, 'Very much better in every way'
162	Q.A.	F.	22	Gastric and R. iliac fossa	2 years dyspepsia, pain 6 months	Colopexy. June 13	Writes, Oct., 1919, 'Quite fit in health; I have had no trouble since operation'
163	C.J.	M.	29	Gastric	Many years dyspepsia, very severe last 18 months	Appendix removed. Colopexy. June 30	Dec., 1919, 'Much better, only slight flatulence'
164	A.A.	F.	31	Gastric	3 years dyspepsia, pain about 6 months. Constipation last 2 years	Simple appendix removed. Colopexy. July 23. Retroperitoneal small intestine in this case	Dec., 1919, 'Slow improvement'
165	T.H.	M.	36	Gastric	Several years dyspepsia, pain and loss of weight 2 years	Simple appendix removed. Colopexy. July 28.	Dec., 1919, 'Reports that he is very pleased with himself'
166	M.C.	F.	41	Gastric and R. iliac fossa	Several years indigestion, much debility	Simple appendix removed. Colopexy. Aug. 14	Dec., 1919, 'I am steadily improving'
167	E.M.	F.	46	Gastric	10 years dyspepsia and pain. Appendix removed 5 years ago	Colopexy. June 14	Dec., 1919, 'Has put on 1 stone in weight, but is still constipated'
168	E.G.	F.	35	Gastric	10–12 years chronic dyspepsia and pain. Appendix removed 4 years ago, no relief	Colopexy. Aug 6.	Dec., 1919, 'Condition much improved'
169	M.I.	F.	25	Duodenal	5 years dyspepsia and hunger pain, appendix removed 2 years ago, no relief	Colopexy. Aug 7	Oct. 26, 1919. 'No pain or indigestion since operation'
170	M.W.	F.	31	Gastric	10 years dyspepsia, 2 years pain, 1 year complete invalid	Simple appendix removed. Colopexy. Oct. 20	
171	H.W.	F.	29	Gastric	Dyspepsia since about 15, pain on and off last 4 years	Simple appendix removed. Colopexy. Oct. 19	
172	S.T.	F.	51	Gastric and biliary	Six months gastric pain following a biliary crisis, gall-bladder still large and tender	Simple appendix removed. Band from ventral aspect of gall-bladder to hepatic flexure cut	

171	H.W.	F.	29	Gastric	Dyspepsia since about 15, pain on and off last 4 years	Simple appendix removed. Colopexy. Oct. 19	
172	S.T.	F.	51	Gastric and biliary	Six months gastric pain following a biliary crisis, gall-bladder still large and tender	Simple appendix removed. Band from ventral aspect of gall-bladder to hepatic flexure cut away. No stones. Colopexy. Nov. 7	
173	G.B.	F.	29	Gastric and R. iliac fossa	Weak inside since childhood, dyspepsia many years, pain last 2 years	Simple appendix removed. Colopexy. Nov. 12	
174	D.F.	F.	31	Gastric	Ten years pain and dyspepsia, prolonged treatment as 'gastric neurosis,' bad migraine, much emaciation	Simple appendix removed. Colopexy. Nov. 14	
175	G.P.	M.	37	Gastric	Dyspepsia 12 years, bad attacks of erratic stomach pain	Simple appendix removed. Jackson's membrane divided. Colopexy. Nov. 22	
176	A.P.	F.	42	Gastric and R. iliac fossa	'Many years dyspepsia', attacks of diarrhœa alternating with constipation, frontal migraine	Simple appendix removed. Colopexy. Nov. 24	
177	G.A.	F.	54	Gastric and renal	30 years dyspepsia, always under treatment, renal crises 1 year, only relieved by hyoscine	Simple appendix removed. Colopexy. Dec. 2	
178	de G.	F.	29	Gastric	Always an invalid, and constipated from childhood, gastric pain and regurgitation, profound emaciation	Diseased appendix removed. Colopexy. Dec. 5	

ADDENDA.

179	M.T.	F.	25	R. iliac fossa and gastric	8 years dyspepsia and dragging pain	Simple appendix removed. Colopexy. Sept. 5, 1917	Writes, Nov., 1919, 'Ever so much better. I have lost all pain and discomfort, mild aperients still necessary
180	A.S.	F.	37	Duodenal	Pain and dyspepsia since childhood, appendix removed 5 years ago; no better	Colopexy. June 5, 1918	Dec., 1919, 'Temporary relief, and now bad gastric pain; operation a failure'

ON THE RESULTS OF BRIDGING GAPS IN INJURED NERVE TRUNKS BY AUTOGENOUS FASCIAL TUBULIZATION AND AUTOGENOUS NERVE GRAFTS.

By HARRY PLATT, Manchester.

It is now a surgical commonplace that the primary destructive effects of the missiles of modern warfare, the secondary slow destruction caused by prolonged suppuration, and the massive production of dense scar tissue, have together rendered the operative repair of injuries of peripheral nerve trunks an exceedingly difficult feat in many cases. Where end-to-end suture has been considered mechanically impossible, many alternative procedures have been recommended and practised during the past five years. At the present time all methods of bridging gaps in injured nerve trunks have fallen into disrepute, but it cannot yet be said that universal condemnation of such procedures is the rule. In particular, the use of autogenous, or even homogenous, nerve grafts is still considered by many to be a justifiable and satisfactory method of repair. In this paper is presented a study of the results of a series of operations in which autogenous nerve grafts combined with fascial tubulization, and tubulization alone, were used. In a total of 430 peripheral nerve operations performed in my services between March 1, 1915, and Oct. 31, 1919, 46 explorations revealed complete lesions in which end-to-end suture was considered at the time to be impossible of attainment. In 15 instances the gap in the injured nerve was bridged by a combination of an autogenous nerve graft and fascial tubulization, in 10 cases by fascial tubulization alone, and in 1 case by an autogenous vein graft: 26 operations in all.

Table of Nerves in which the Bridge Operations were Employed.

Nerve	Fascial Tubulization and Nerve Graft	Fascial Tubulization alone	Vein Tubulization
Ulnar	6	4	—
Median	2	4	—
Musculospiral ..	3	1	1
Posterior interosseous	1	1	—
Sciatic	2	—	—
Posterior tibial ..	1	—	—
Totals	15	10	1

Total number of operations, 26.

It is interesting to note that 20 of these bridge operations were performed prior to December, 1917, out of a total of 106 nerve operations up to that date. These operations were based on the reputed value of both the nerve graft and fascial sheath at that period; and furthermore, in all my early operations a conservative attitude was deliberately adopted towards extensive freeing of nerve trunks, in view of the potential risks of endangering the vascular supply of the nerves explored. This high percentage of apparent failures to achieve end-to-end approximation is thus explained. The justification for the use of nerve grafts rested largely on isolated statements in the pre-war literature of peripheral nerve surgery, as it must be admitted that detailed and accurate end-results were not available. The use of fascial tubulization had a much more scientific basis, as seen

by the results obtained in experimental nerve surgery in dogs by Dean, Lewis, and Kirk.[1] These observers, in a preliminary communication in 1915, and in a further elaborate study published in 1916, established beyond all doubt the occurrence of regeneration of axis cylinders across considerable gaps in nerve trunks in dogs; this regeneration proceeded in an orderly fashion from the proximal to the distal ends of the nerve trunks inside autogenous fascial tubes. In no experiment was collapse or obliteration of the lumen of the fascial tunnel noted. These results are undoubtedly suggestive of the possibility of similar phenomena occurring in human peripheral nerves.

In all my operations the fascial tube was regarded as the essential conducting tunnel, even when combined with a nerve graft, as it was illogical to suppose that a small cutaneous nerve could possibly conduct the axons of the total cross-section of the larger injured nerve.

Operative Technique.—The nerve grafts used were single segments of the radial, internal cutaneous, musculocutaneous, internal saphenous, and internal plantar nerves. The graft in every case was of considerably smaller calibre than the recipient nerve, and it was sutured to the cross-section of the proximal and distal ends with fine catgut. The whole area was surrounded by an autogenous fascial sheath, in most cases obtained from the fascia lata of the thigh. This tube, after closure, was distended with sterile olive oil, or its interior smeared with sterile vaseline. Where the nerve graft was omitted, the ends of the injured nerve trunk were connected inside the fascial tube by two fine catgut strands.

It has been possible to follow up 18 cases in which the above operations were performed, over varying periods of time. Furthermore, in four instances re-exploration of the nerve at a later date provided electrical, macroscopical, and histological information of considerable value. In the post-operative study of these cases, my colleague, Professor J. S. B. Stopford, has taken the major share. I am indebted to him for free access to his detailed records of all peripheral nerve injuries seen in my services, and for his collaboration, which has necessitated almost daily association during the past three years.

The histological investigations on the material provided by the secondary operations are at the present time incomplete, but will be made the subject of a future communication by Professor Stopford.

REPORTS OF CASES.

Group A.—FASCIAL TUBULIZATION COMBINED WITH NERVE GRAFT.

Case 1.—**Musculospiral nerve injury, complete, lower third of the left arm.**

H. W. (civilian). Wounded Oct., 1916. Compound fracture left humerus; severe laceration of soft tissues in region of elbow; fractured radius and ulna; resulting bony ankylosis of elbow at right angles.

Operation, June 5, 1917.—Complete destruction of nerve trunk, with 2½-in. gap; ankylosis of elbow prevented end-to-end approximation of the nerve; radial nerve graft and fascial sheath inserted.

After-treatment.—Regular; examined repeatedly. July 6, 1918.—No sign of motor or sensory recovery; no reaction to galvanism in any muscle of the extensor group. July 13, 1918.—Tendon transplantation performed. Feb. 13, 1919.—Function of the tendon transplantation is good; is now working. No alteration in the nerve syndrome. Aug. 6, 1919.—Functional capacity afforded by tendon transplantation is maintained. No change in the nerve syndrome.

Result of Operation.—No sign of regeneration after twenty-six months.

Case 2.—**Musculospiral nerve injury, complete, middle of upper arm.**

Pensr. A. Wounded July 1, 1916. Compound fracture of humerus, extensive destruction of triceps muscle, and prolonged suppuration.

Operation, July 14, 1917.—The nerve trunk in the region of the lesion was represented by a fibrous cord 4 in. in length. Radial nerve graft and fascial sheath inserted.

After-treatment.—Regular; seen repeatedly for examination. Feb. 28, 1919.—No sign of regeneration, no galvanic response in extensors. Sept. 25, 1919.—No sign of regeneration. Condition as before. Oct. 4, 1919.—Tendon transplantation performed.

Result of Operation.—No regeneration after twenty-six months.

Case 3.—**Complete median nerve injury, upper third upper arm.**

Pte. B. Wounded Aug. 4, 1916. Compound fracture of humerus; gross atrophy of upper arm, fibrosis of the biceps.

Operation, June 12, 1917.—In the region of the scarring the median nerve was represented by a fibrous cord 3 in. in length. Internal cutaneous nerve graft and fascial sheath inserted.

After-treatment.—Regular; examined repeatedly. Six months after the operation there was an apparent action in the flexor longus pollicis, but this was later proved to be a trick movement. Ten months after the operation an apparent diminution of the area of anæsthesia in the median area was noted. March 8, 1919.—No sign of motor recovery; no galvanic response in any median muscles; Tinel's sign absent. There is a palpable bulb on the nerve trunk proximal to the graft. The patient has developed a trick movement which closely simulates action in the palmaris longus. There is also apparent voluntary power in the flexor sublimis digitorum. Re-exploration advised.

Re-exploration Median Nerve, April 30, 1919.—Exsection of the grafted area, end-to-end suture. The median nerve trunk was found to terminate above in a large bulb which was adherent to the adjoining remains of the coracobrachialis muscle; running down from the bulb was a narrow flattened cord, which was recognizable as composed of the original fascial sheath. When traced downwards this fine cord became continuous with the distal part of the nerve trunk, which was only half the calibre of the nerve trunk above the level of the graft. Faradic stimulation of the nerve trunk above and below the grafted area, and of the area itself, gave absolutely no response in any muscle in the forearm or hand. The grafted area was resected, the line of section passing above the proximal bulb and through the normal nerve trunk below. On the cross-sections the nerve bundles were seen to be of the gelatinous type. End-to-end suture was accomplished, with the elbow in acute flexion and the arm adducted to the side. May 9, 1919.—Motor and sensory phenomena precisely as before operation; the trick movement in the palmaris longus and the apparent activity of the sublimis digitorum is still present.

Result of Nerve-graft Operation.—No sign of regeneration twenty-one months after operation.

Case 4.—**Ulnar nerve injury, left forearm.**

L.-Cpl. D. Wounded July 1, 1916.

Operation, Dec. 20, 1916.—Gap of 2½ in. revealed; musculocutaneous nerve graft and fascial sheath inserted.

After-treatment.—Regular. Lost sight of patient for a considerable period. Feb. 13, 1919.—Full motor and sensory loss; Tinel's sign absent.

Result of Operation.—No sign of motor, sensory, or sympathetic recovery after twenty-six months.

Case 5.—**Musculospiral nerve injury (left).**

Pte. H. Wounded March 27, 1916. Compound fracture of the humerus, with necrosis; prolonged suppuration; extensive destruction of the triceps.

Operation, March 21, 1917.—The musculospiral nerve was completely obliterated over an extent of 5 in. Radial graft and fascial sheath inserted.

After-treatment.—Regular until February, 1918; examined repeatedly. Feb. 28, 1919.—Complete paralysis of the extensor group; sensory loss as before operation; Tinel's sign absent.

Result of Operation.—No sign of regeneration, motor or sensory, after twenty-three months. Tendon transplantation advised, but refused.

Case 6.—**Ulnar nerve injury, left forearm.**

Pte. H. Wounded Sept. 9, 1916. Non-union of fracture of the ulna.

Operation, April 3, 1917.—Gap 2½ in. in nerve before trimming; radial graft and fascial sheath inserted.

After-treatment.—Regular; examined repeatedly. April 21, 1919—Full motor and sensory loss; vasomotor paralysis as before; Tinel's sign absent.

Result of Operation.—No sign of motor, sensory, or sympathetic recovery after two years. Re-exploration advised, but refused.

Case 7.—**Median nerve injury, right forearm.**

Pte. M. Wounded Sept. 26, 1916. Compound fracture of the radius, with non-union; extensive muscle and tendon destruction, with matting and contractures.

Operation, May 30, 1917.—Six-inch gap in the trunk was revealed; this was bridged by a musculocutaneous graft and a fascial sheath.

After-treatment.—Regular. Fourteen months after the operation there was an apparent action in the abductor pollicis. The thenar eminence showed practically no flattening; no sensory recovery. Feb. 10, 1919.—Full median sensory loss, with vasomotor paralysis; no Tinel's sign; apparent action of all muscles in thenar group; (? abnormal nerve supply). Sept. 5.—Condition as before.

Result of Operation.—No true sign of regeneration, motor, sensory, or sympathetic, after twenty-six months. The ununited fracture of the radius has since been treated by bone-shortening of the ulna. Re-exploration of the grafted median nerve is contemplated.

Case 8.—**Ulnar nerve injury, right forearm.**

Pte. R. Wounded April 9, 1917. A complete lesion of the median nerve co-existed with extensive laceration of the bellies of the flexor muscles.

Operation, Aug. 29, 1917.—End-to-end suture of median nerve: ulnar nerve showed extensive destruction of the trunk; 3½-in. gap; musculocutaneous graft and fascial sheath inserted.

After-treatment.—Regular; examined repeatedly. The median nerve showed steady signs of regeneration, and at the end of eighteen months there was recovery in the thenar muscles and slight residual analgesia. The ulnar nerve syndrome remained unchanged, and at the end of eighteen months there was a complete absence of any regenerative sign.

Re-exploration of Ulnar Nerve.—The grafted area was exsected, and end-to-end suture of the nerve performed after displacement in front of the internal epicondyle.

Description of the Grafted Area.—This appears as a soft collapsible tube, the fascia being unchanged in physical appearance and presenting few adhesions to the surrounding tissues. The graft could not be felt inside the tube; on splitting the tube along the original line of its closure, it was found that the lumen was almost completely obliterated, and, to the naked eye, there was no sign of the nerve graft.

Description of Microscopical Section (Professor J. S. B. Stopford).—The proximal end shows a neuroma formation; in the centre of the fascia lata are seen a few bundles of wavy fibres occupying the position of the graft; no fibres seem to have reached the distal segment of the nerve.

Result of Operation.—No sign of ulnar regeneration after eighteen months.

Case 9.—**Ulnar nerve injury in the region of the left elbow.**

Cpl. D. Ankylosis of elbow-joint.

Operation, May 18, 1917.—Gap of 2½ in. revealed; end-to-end suture was prevented by the fixation of the elbow-joint. Internal cutaneous nerve graft inserted with fascial sheath.

After-treatment.—The patient had regular treatment at Shepherd's Bush Special Military Surgical Hospital, and was discharged at the end of twelve months with no sign of any recovery in the ulnar nerve.

Result of Operation.—Complete absence of any signs of regeneration after twelve months.

Case 10.—**Posterior interosseous nerve injury.**

Cpl. R. Wounded Sept., 1916. Bony ankylosis of elbow-joint.

Operation, March 17, 1917.—Gap of 2½ in. in the posterior interosseous nerve; radial nerve graft and fascial sheath inserted.

After-treatment.—Irregular. March 18, 1919.—No sign of recovery in any muscles of the extensor group. No response to faradism; profound wasting of the extensor bellies. Tendon transplantation advised; this operation was performed; functional result good.

Result of Operation.—Complete absence of signs of regeneration after two years.

Case 11.—**Sciatic nerve injury (left).**

Pte. C. Wounded Nov. 6, 1915. Compound fracture of femur; complete ankylosis of knee-joint; extensive destruction of posterior thigh muscles.

Operation, March 8, 1918.—Gap of 6 in. in the sciatic nerve: lesion quite irreparable. Insertion of internal saphenous nerve graft and fascial sheath. March 8, 1919.—Complete absence of any signs of regeneration.

Result of Operation.—No signs of regeneration after one year.

Group B.—FASCIAL TUBULIZATION OPERATIONS.

Case 12.—**Musculospiral nerve injury.**

Pensr. B. Wounded Aug. 3, 1916. Compound fracture of the left humerus, with extensive destruction of muscles.

Operation, Nov. 3, 1917.—Destruction of musculospiral over a length of 5 in.; gap bridged by fascial sheath and catgut strands.

After-treatment.—Irregular; but seen repeatedly for examination.

Result of Operation.—No sign of motor or sensory recovery after fourteen months. Extensor bellies practically disappeared. Tendon transplantation advised.

Case 13.—**Ulnar nerve injury, right forearm.**

Bdr. D. Wounded April 15, 1917.

Operation, Nov. 16, 1917.—Gap of 3 in. found; fascial sheath and catgut strands inserted.

After-treatment.—Regular.

Result of Operation.—No sign of motor, sensory, or sympathetic recovery after fourteen months.

Case 14.—**Median nerve injury, right forearm.**

Pensr. H. Compound fracture of radius and ulna, with considerable destruction and matting of flexor bellies.

Operation, July 9, 1918.—After extensive freeing of the median nerve there was a 2-in. gap, even with the elbow in acute flexion. The gap was bridged by a fascial sheath and catgut strands as a temporary measure. March 14, 1919.—No sign of regeneration.

OPERATION, March 28, 1919.—Exsection of the bridged region, end-to-end suture. At the second operation the fascial sheath was found to be a collapsed tube showing complete obliteration of its lumen. The nerve trunk above the sheath was indurated, and presented a slight bulbous thickening; a corresponding appearance was seen at the distal extremity of the sheath. End-to-end suture was obtained, after extensive freeing of the nerve trunk, with the wrist and elbow acutely flexed.

RESULT OF OPERATION.—No sign of regeneration after eight months.

Case 15.—**Ulnar nerve injury, left forearm.**

Pensr. B. Compound fracture of the radius, July 13, 1916, with non-union.

OPERATION, May 21, 1918.—After exsection of 2½ in. of fibrosed nerve trunk, a gap of ¾ in. was left. This was bridged by a fascial sheath and catgut strands as a temporary measure. March 1, 1919.—Tinel's sign absent; ? recession of analgesia; no motor recovery.

RESULT OF OPERATION.—No definite sign of regeneration after nine months. Re-exploration of the nerve was postponed owing to the patient's poor general health.

Case 16.—**Median nerve injury, right forearm.**

Pensr. C. Wounded July 11, 1915. Co-existing complete lesion of the ulnar nerve and matting of the flexor tendons and bellies; previous ligature of the brachial artery.

OPERATION.—July 21, 1918.—End-to-end suture of the ulnar nerve after anterior displacement. Median nerve, 3-in. gap bridged by sheath and catgut strands. No alteration in median syndrome after four months.

RE-EXPLORATION OF MEDIAN NERVE, Dec. 13, 1918.—The bridged area was exsected, and end-to-end suture obtained with ease. The segment removed consisted of a dense fibrous cord closely resembling a nerve trunk. The fat on the fascial sheath was still recognizable; no traces of catgut.

HISTOLOGICAL EXAMINATION.—Obliteration of the lumen of the fascial sheath, which is filled with young fibrous tissue, but in which there are no traces of any nerve fibres.

RESULT OF OPERATION.—No sign of regeneration after four months. Histological appearances of bridge suggest that no regeneration could have occurred even after a more prolonged period of waiting.

Case 17.—**Median and ulnar nerve injuries in region of right elbow.**

Pte. D. Ankylosis of the elbow in right-angle position; considerable destruction of the soft tissues.

OPERATION, Sept. 4, 1917.—2½ in. in both nerves bridged by fascial sheaths and catgut strands. Feb. 27, 1919.—No sign of any motor recovery; reaction of degeneration in muscles supplied by median and ulnar nerves; full sensory loss.

RESULT OF OPERATION.—No sign of regeneration after seventeen months.

Class C.—AUTOGENOUS VEIN TUBULIZATION.

Case 18.—**Musculospiral nerve injury.**

Pte. H. Wounded July 1, 1916. Compound fracture of the humerus, with extensive destruction of the triceps muscles.

OPERATION, March 17, 1917.—Musculospiral nerve was destroyed over a length of 6 in. Cephalic vein inserted into the gap as an autogenous tube. Feb. 27, 1919.—No sign of recovery.

RESULT OF OPERATION.—Failure of regeneration after twenty-three months.

RESULTS OF OPERATIONS.

Of the eighteen cases in the above series, in only three was the period of time after the operation under twelve months. Ten were examined eighteen months or more after the operation, and six of these after a two-year period. The result in every case was identical, viz., a complete absence of any clinical sign of recovery. This, in my opinion, is sound evidence in favour of regarding the operative procedures as permanent failures, but in itself is not enough. It may be suggested that the process of regeneration across gaps in nerve trunks, if physiologically possible, must be of necessity slow and, from the point of view of topography, inaccurate. Signs of recovery, therefore, would be long delayed. On the other hand, the re-exploration operations, particularly in two of the cases in which observations had been limited to a period of less than a year, have afforded evidence in favour of the permanency of the absence of regeneration. When a complete histological study of the tissues resected at secondary operations is available, I consider that further convincing evidence will be forthcoming as to the futility of the operations in question.

It will be noted that in two cases, Nos. 3 and 7, some months after the operation

there developed a change in the nerve syndrome. In *Case* 3 the alterations were striking, and at first were regarded, after serious consideration, as indicative of regeneration. In this patient, after seven months there appeared an apparent action in the flexor longus pollicis ; shortly after, the area of analgesia underwent an apparent diminution. Some time before the nerve was submitted to a secondary exploration there appeared what seemed to be a definite voluntary action in both the palmaris longus and flexor sublimis digitorum. With the possibility of 'trick' movements in mind, but with no definite proof of this, the median nerve was re-explored, and was found to be silent to direct faradic stimulation. Stimulation of the ulnar nerve in this region produced the movements investigated before the operation. In this patient there was an intractable contracture of the flexor bellies, and the activity of the flexor profundus digitorum produced in the relatively immobile fingers the movements characteristic of the sublimis muscle.

In *Case* 7 the change in the syndrome was purely motor ; after fourteen months the thenar eminence, previously flattened, showed a definite increase in volume, with an apparent recovery in the abductor pollicis muscle. In complete median nerve injuries one is accustomed to see excellent function and development in the flexor brevis pollicis belly so frequently, that one looks upon this muscle as supplied by the ulnar nerve in a large percentage of cases. In these cases, however, there is always a narrow zone of flattening along the radial border of the metacarpal in the position of the abductor. In the case under consideration this localized wasting of the abductor pollicis was conspicuously absent. Owing to the lack of any sensory change after twenty-six months, this apparent recovery in the abductor pollicis muscle is not considered indicative of true regeneration. At the time of writing, a secondary exploration of the nerve has not been carried out. Until this is done, the motor phenomena in this case cannot be explained with any certainty.

There can be little doubt that many so-called successful nerve-graft and fascial-bridge operations have been regarded as such owing to the development of signs of alteration in the motor syndrome, as manifested by muscle actions, which are purely trick movements. The path of the clinical observer in peripheral nerve injuries is beset with pitfalls. It is for this reason that the findings of exploratory operations, both primary and secondary, must be described with meticulous accuracy, particularly with regard to the results of direct faradic stimulation of the nerve trunks, motor branches, and muscle bellies exposed in the field of operation. The histology of any tissue resected completes an adequate conception of the morbid anatomy and physiology of an injured peripheral nerve.

CONCLUSIONS.

1. In eighteen operations in which fascial tubulization combined with autogenous nerve grafts, fascial tubulization alone, and autogenous vein tubulization (one case), were used, there was a complete absence of any clinical sign of recovery. The shortest period over which observations were made was four months, the longest period twenty-six months.

2. Secondary exploration in four cases showed complete silence of the nerve trunk to direct faradic stimulation. End-to-end suture was accomplished in all after exsection of the bridged segment.

3. At the re-exploration operations, partial or complete obliteration of the lumen of the fascial tube was noted.

4. In two specimens examined histologically, one, a tubulization alone, showed obliteration of the lumen of the tube by fibrous tissue in which no nerve fibres could be found. In the second, a graft and tubulization combined, nerve fibres were present in the centre of the obliterated tubule eighteen months after the operation. There was no sign of continuity between the proximal and distal ends through this strand of nerve fibres.

5. The early re-exploration of all graft and fascial-bridge operations is advisable.

REFERENCE.

[1] Dean, Lewis, and Kirk, *Jour. Amer. Med. Assoc.*, 1915, lxv ; *Trans. Amer. Surg. Assoc.*, 1916, p. 486.

EXCISION OF A SUBCLAVIAN ANEURYSM.

By L. R. BRAITHWAITE, Leeds.

In 1898 Sir Berkeley Moynihan[1] published a case of subclavian aneurysm treated by complete excision, and claimed for excision as against ligature: (1) A lower mortality (based on Delbet's statistics[2]); (2) An absence of recurrence; (3) A lower percentage of cases of gangrene; and (4) A higher 'quality of recovery.'

Cases of excision of subclavian aneurysm will be found to be exceedingly rare. Schopf, of Vienna,[3] has recorded a successful case of excision of a 'subclavio-axillary' aneurysm, but as the ligature was applied to the proximal vessel 'from the axilla under the clavicle,' the aneurysm was probably more axillary than subclavian. Halsted, in America,[4] records what is probably the best case of excision of a subclavian aneurysm, the proximal vessel being ligatured in the first part, and complete recovery of the patient resulting. In this case "the greater part of the clavicle, a piece of the deltoid muscle, and about six centimetres of the subclavio-axillary vein were removed with the aneurysm."

Moynihan's case was attacked by a novel method, the greater part of the clavicle being excised and turned down, with its adherent pectoral muscle. Later it was wired back into position. In this case death resulted on the fifty-ninth day after operation, and was due to the development and rupture of a second aneurysm proximal to the one excised, the whole of the vessel wall being the seat of patchy degeneration.

A careful search in the literature on subclavian aneurysm has revealed no other case of excision. It is this fact which decided the author to publish the case which follows.

During the Great War, penetrating and perforating injuries of the subclavian vessels have been fairly common (28 cases are recorded[5]), nearly all involving artery and vein. When an aneurysm resulted, as happened in 24 cases, it was purely arterial in respect of 13, and in 11 was arteriovenous. Unfortunately, nearly all were lost sight of, but in the few in which an attempted excision was made a fatal issue resulted on the table in every case.

Complications due to extravasation of blood and to infection, with their resulting obliteration of all the normal anatomical details in the vicinity, as well as the almost immediate and severe hæmorrhage following manipulation of what was always a false sac, made operative interference exceedingly dangerous. It was only undertaken when a rapid increase in the size of the swelling made it imperative.

In two cases where the author acted as assistant these difficulties were so great, despite removal of the clavicle and the upper angle of the manubrium sterni, that the exact origin of the hæmorrhage was not discovered until after death. It would be unreasonable to include such cases in a discussion on the treatment of true aneurysm of the subclavian artery, but it is thought that the republication of a method which has given some measure of success may help in the treatment of aneurysms of this vessel which may yet arise as the result of war injuries.

Sir George Makins,[5] speaking of arteriovenous aneurysm, says: "Is it better to divide the clavicle or not? In the majority of cases, unless the junction of the subclavian and axillary arteries requires to be exposed, it is quite unnecessary if the sternomastoid be divided; section of the bone increases the severity of the operation, and entails risk of injury to the aneurysmal sac or the veins. I think the procedure should be reserved for cases of exceptional difficulty, and rarely resorted to. My own experience leads me to regard operation for arteriovenous aneurysms in this region as the most difficult and dangerous of any that can be undertaken."

Author's Case. —T. J., age 46, a miner, admitted Nov. 29, 1913. Three months previous to admission to the General Infirmary, Leeds, he noticed some pain in the region of the right shoulder, shooting down the arm. He thought it was rheumatism, and as it did not improve he saw his doctor, who treated him by local applications, but without relief. Two months before admission he noticed a lump over the collar bone, saw his doctor again, and was eventually sent to the Infirmary, where the condition was diagnosed as an aneurysm of the subclavian artery.

After admission he said the pain was of an aching character, shooting down to the thumb and three outer fingers; it was always present, but was easier when he lay down. As the pain grew less in intensity it left a numbness in the hand and fingers.

EXAMINATION.—There was a rounded swelling about the size of a hen's egg above and behind the middle third of the right clavicle in the position of the subclavian artery. The skin was not adherent to it, and it was not tender except on deep pressure. There was an expansile pulsation synchronizing with the heart-beat, and over it a loud systolic murmur. Both carotid and radial pulses were equal on the two sides. There was no history of syphilis or of trauma. Wassermann

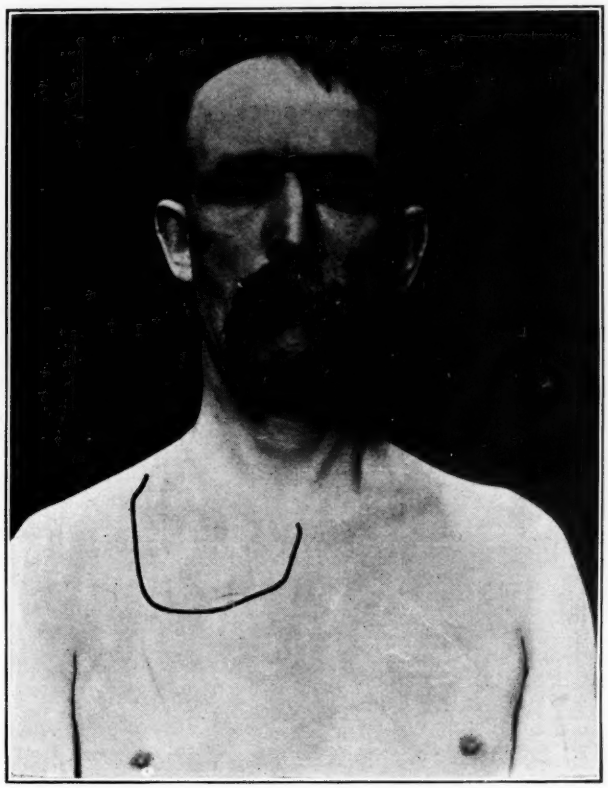

FIG. 362.—The curved incision for excision of a subclavian aneurysm.

test was positive, and for three weeks before operation he was given potassium iodide in 20-gr. doses three times a day.

OPERATION, Dec. 22, 1913.—The operation was planned exactly on the lines recommended by Moynihan.[1] Ether was administered by Dr. H. J. Macvean, the patient being flat on his back. The area of operation was sequestrated by screen, and the skin very carefully prepared by the biniodide spirit and Harrington method. Mr. E. R. Flint very ably assisted with the operation. A curved incision with its convexity downwards was made over and below the clavicle (*Fig.* 362), and the flap of skin and platysma turned upwards, exposing the clavicle freely; the external jugular vein was divided and turned up in the flap. Most of the clavicular head of the sterno-mastoid was divided. Preliminary holes were bored through the clavicle for the subsequent suture, and it was then divided in two places, turning down rather more than the middle half of it on to the chest, together with the attached portion of the pectoralis major muscle (*Fig.* 363). The omohyoid, reduced to a film of muscle fibres, was cut away, and a very full exposure of the whole area obtained. The extent of the aneurysm was then evident, the tumour extended outwards beyond the limits of the wound, and inwards it appeared to merge into the scalenus anticus

muscle, which apparently became inserted into it. Upwards it was limited by the brachial plexus, which was firmly adherent to it. Curling over the face of the tumour were two very big arteries, the transversalis colli above and the suprascapular below; these were carefully stripped off and retracted, the former upwards, the latter towards the clavicle. Several small veins were divided and removed. The internal jugular and subclavian veins were very prominent though never in the way, whilst the phrenic nerve was easily seen and avoided.

It was very striking at this stage to notice the lack of definition of the tumour above and to the inner side compared with its sharp line of demarcation from the subclavian vein below. (*Fig.* 363).

The scalenus anticus was now defined, after dividing the dense bands of fibrous tissue which passed from it to the sac. The tubercle on the first rib was easily felt, and it was found that the sac extended for about one-third of an inch into the second part of the vessel; its limit could be seen by strongly retracting the scalenus anticus inwards. The brachial plexus presented great difficulties, the wall of the sac being very thin at this part and most firmly adherent. Having cleaned the sac as well as possible, an attempt was made to pass an aneurysm needle round the sac at the level of the first rib; the intention being to slide a ligature from the body of the sac

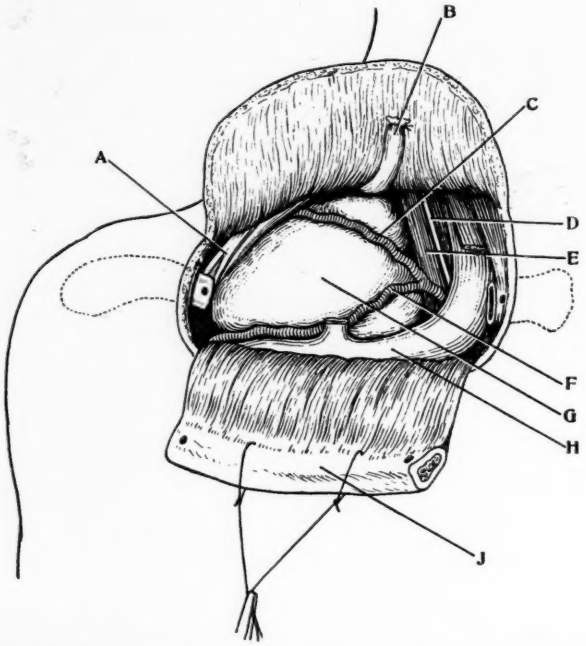

FIG. 363.—Excision of subclavian aneurysm. (A) Brachial plexus; (B) External jugular vein; (C) Transversalis colli artery; (D) Phrenic nerve; (E) Scalenus anticus; (F) Suprascapular artery; (G) Aneurysmal sac; (H) Subclavian vein; (J) Clavicle (turned down).

inwards under the scalenus anticus, and so on to the proximal portion of the second part of the artery beyond the sac. The scalenus anticus was retracted until the carotid artery was seen, and the needle passed from before backwards. On reaching the rib there was a very smart spurt of arterial blood, which ceased on withdrawal of the needle.

The supposition that the sac had been perforated was confirmed when, on passing the forefinger down along the needle track, there was a very fierce and copious escape of blood, and the finger passed into the sac of the aneurysm, which at this point was eroding the rib, the roughened surface of which could be easily felt.

Fortunately the left forefinger had been introduced (the operator is right-handed), and, as any attempt to withdraw the finger was followed by the most alarming hæmorrhage, it was pushed deeper in, blocking the lumen of the proximal vessel, whilst with the right hand a Moynihan's cholecystectomy forceps, with a short curved beak, was applied to the second part of the vessel at the inner border of the first rib.

With the hæmorrhage thus controlled, the left forefinger being still within the sac, the assistant rapidly cleared the outer margin of the sac, exposing the efferent vessel and dividing it between two stout catgut ligatures half an inch beyond the sac.

The whole sac was now turned upwards and inwards, a kangaroo tendon ligature was applied to the subclavian artery just proximal to the cholecystectomy forceps, a second ligature

of stout catgut was applied in the groove left by removal of the forceps, and the sac was then removed.

The area was now completely dry ; the clavicle was drawn up, and sutured in position with chromic catgut, the sternomastoid was replaced, and the wound closed, leaving a small tube drain at the outer angle.

Subsequent History.—There was no shock, and, although the arm was a little cold and blue for a day, the patient never had a bad symptom or sign. The wound healed by first intention without any suppuration. He was up in three weeks, the pain had completely vanished, and

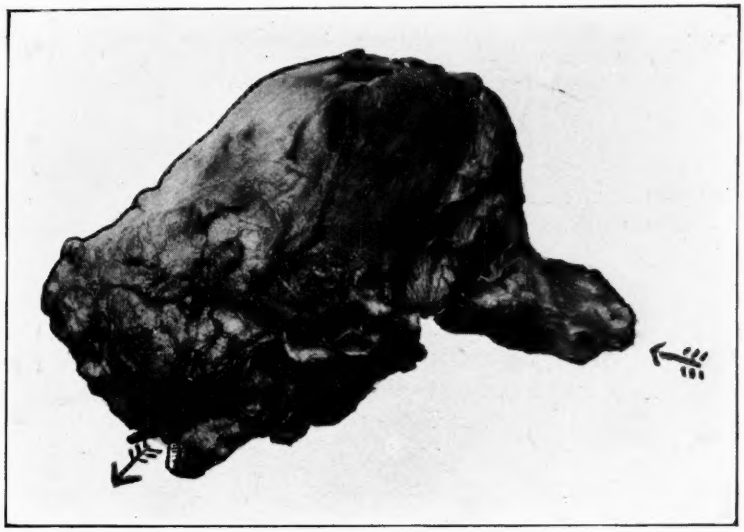

FIG. 364.—Subclavian aneurysm.

he was discharged from the hospital on Jan. 17, 1914. When seen again, in June, 1914, he was very well indeed ; there was no pain of any kind, sensation in the hand was normal, and there was no difference in the radial pulses. He returned to 'surface' work at the pit during the summer of 1914, and in 1919 had worked at the coal face. The only drawback, in what may otherwise be considered a perfect result, is that the portion of clavicle which was excised did not unite by bony union, but is movable in false joints.

There is no recurrence of the aneurysm.

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LOCAL DISCOLORATION OF THE ABDOMINAL WALL AS A SIGN OF ACUTE PANCREATITIS.

By G. GREY TURNER, Newcastle-on-Tyne.

In 1912 I was asked to see a big stout woman, 54 years of age, suffering from an acute abdominal illness of three days' duration. I was at once struck by the appearance of the abdominal wall surrounding the umbilicus. In this neighbourhood there was an area of discoloration about 6 inches in diameter, of a bluish colour, very like the early post-mortem staining seen on the abdominal wall, or the appearance of the skin in a late case of extravasation of urine. The area was slightly raised, and pitted on pressure. In this case (Reg. No. 5323) the patient suffered from acute pancreatitis, with much effusion into the peritoneal cavity. She lived nine days after operation, and the post-mortem examination disclosed a sloughing pancreas with much fat necrosis.

I did not observe this sign again until September, 1917, when an old soldier (Reg. No. 9536), 53 years of age, tall and of spare build, came under my observation. He had previously suffered from attacks of abdominal pain, but they had always been of short duration. On Sept. 17 he had a seizure like his old attacks, but so severe that it caused

Fig. 365.—Photograph of a case of acute pancreatitis, showing the area of discoloration referred to in the text.

profuse clammy perspiration, and was only partially relieved by ¾ gr. of morphine hypodermically. Next day the pain was easier, but there was some tenderness over the gall-bladder region. On the 19th the abdomen was beginning to be distended. On the 20th the pain was still better, but there was more distention, and the pulse was now over 100. The attack was recognized as being much more severe than any from which he had previously suffered. On the evening of this day I first saw the patient. He looked very ill and distinctly toxic. The tongue was dry, the pulse 112, and the temperature over 100°. The abdomen was generally distended, and he was very tender and rigid in the right hypochondrium. No mass could be felt, and no superficial discoloration was noticed, although it was not especially looked for. The next day the patient was more comfortable generally: the pulse was still 112, though the temperature had come down, and he had passed considerable flatus. The tenderness over the gall-bladder region was very marked, and I now noticed two large discoloured areas in the loins. They were about the size of the palm of the hand, slightly raised above the surface, and of a dirty-greenish

colour. There was a little œdema, with pitting on pressure, but there was no pain or tenderness. The urine was found to be full of sugar. A diagnosis of acute pancreatitis was made; and this was confirmed by an immediate operation. The peritoneal cavity contained a moderate amount of dirty-brownish fluid, and there was a good deal more on passing the fingers down towards the pancreas. There was very extensive fat necrosis of the omentum. The gall-bladder contained large numbers of small calculi. Incisions were made into either loin over the areas of discoloration. The superficial tissues were markedly œdematous, and there was a considerable quantity of thin, dirty fluid from the retroperitoneal space. The subcutaneous and retroperitoneal fat showed areas of necrosis.

The patient stood the operation very well, and for a day or two he improved; but at the end of about a week he began to lose ground, suffering from vomiting, air-hunger, inability to sleep, and steady wasting. Death occurred five weeks from the date of the operation. At the post-mortem examination the pancreas was found converted into a slough, and there was a direct track leading from it to the incisions in either loin. There was also extensive fat necrosis, but the peritonitis had recovered, and the abdominal cavity was clean.

The illustration (*Fig.* 365) is from a photograph taken just before the operation, and accurately represents the situation and size of the patch in the left loin, that on the right side being exactly similar. The condition is clearly due to the direct action of the pancreatic juice, which escapes via the retroperitoneal tissue, and passes by the most direct route to the surface. In the case in which the discoloration was only noted round the umbilicus, the secretion may perhaps have reached that area via the round ligament of the liver.

In a cursory examination of the voluminous literature on pancreatitis I have not observed any mention of this sign.

GASTRIC ULCER: A PLEA FOR GASTRECTOMY.

By E. R. FLINT, Leeds.

WITH A NOTE ON X-RAY DIAGNOSIS

By H. B. SCARGILL, Leeds.

There has been in the past, and still is, so much confusion as to what a gastric ulcer is, that it is necessary, before discussing the subject, to state definitely the condition to which this paper applies. We refer to a localized inflammatory lesion in the stomach, which results in a loss of tissue of at least the full depth of the mucosa, and which, if it heals, does so by fibrous tissue. There is, therefore, a permanent thickening in the stomach wall. This definition excludes the chaps, fissures, and superficial erosions which have swollen the statistics of gastric ulcer. The clinical manifestations of these—digestive disturbance, vomiting, and hæmatemesis—resemble those attributable to gastric ulcer, but there is not destruction of the whole depth of the gastric mucosa, and so they can, and do, heal without scarring.

One would think that, if gastric ulcers were as common as they are reported to be, they, or the scars left after healing, would be met with frequently at operation, or on the post-mortem table, whereas, as a matter of fact, this is not the case. During seven years at the Leeds General Infirmary, 1078 cases of duodenal and gastric ulcers were operated on; only those with visible or palpable evidence were accepted. Of these, 749 were duodenal and 329 gastric, that is, about two and a half of the former to one of the latter. During the same period there were 980 gall-stone operations. The relative frequency of these three conditions was, therefore, six gall-stones to five duodenal ulcers and two gastric ulcers.

The gastric ulcers which come to operation are usually of considerable chronicity, and it might be said that surgeons do not see the acute ulcers. This is undoubtedly true to some extent; but if it is a fact that every ulcer, even an acute one, leaves permanent traces, which I think cannot be denied, and as the scars are so seldom found at operation or post mortem, one is driven to the conclusion that all gastric ulcers are uncommon. The clinical diagnosis of gastric ulcer is so beset with difficulties that it cannot be accepted as proof of the existence of the lesion. Figures compiled from this source show gastric ulcer to be more common than all the cases of gall-stones and duodenal ulcers put together. Moreover, they give a large preponderance of females over males. The reason is that gastric disturbances—especially when associated with hæmatemesis—occurring in young anæmic women are ascribed to ulceration. Such cases, when submitted to operation, show no sign of ulcer according to the definition given. As regards sex, duodenal ulcer is four times more common in men than women, and gastric ulcer occurs in two males to one female.

Causation.—The fundamental factor in the development of gastric ulcer, as in all ulceration, is infection. Rosenow[1] has done some interesting work in this connection. He has found that certain organisms have an elective affinity for the tissues in which they grow, when re-injected into the blood-stream; for example, streptococci can be found in, and isolated from, the base of a gastric ulcer, and, when injected into the blood-stream of another animal of the same species, settle, in about 70 per cent of experiments, in the corresponding organ, and produce a similar lesion. The same applies to the appendix, to the gall-bladder, and in fact to most tissues. This is merely an extension of the observation that pneumococci, for instance, usually choose the lungs for the site of their activities. But Rosenow has gone a step further, and shown that

the same organism, in a different state of virulence, will attack another organ more or less consistently, if kept at this new virulence. He has shown that the streptococcus which ordinarily has an elective affinity for the appendix, will, if the virulence be slightly increased, produce after injection into the blood-stream, not an appendicular lesion, but necrosis of the gastric tissues.

According to Bolton[2] the initial lesions which can give rise to ulcer of the stomach are localized necrosis of the mucous membrane, localized hæmorrhage, and inflammation of the lymphatic follicles of the stomach wall. The common cause of necrosis is bacterial infection, or bacterial toxins circulating in the blood-stream, and, the cells of the gastric mucosa being primarily attacked, the damaged area is destroyed by digestion by the gastric juice. Necrosis may arise in this way without preceding hæmorrhage or lymphatic inflammation; but hæmorrhage is an actual and frequent cause of ulcer, and is likewise due to bacterial toxins circulating in the blood-stream, which, destroying the endothelial cells of the capillaries, pave the way for the local destructive action of the gastric juice. Finally, inflammation of one or more of the lymphatic follicles, so thickly studded along the lesser curvature, may give rise to a submucous abscess which, by rupture into the stomach, allows the juice to act on the base of the ulcer thus exposed. Bolton has also shown that a gastric juice of abnormally high acidity will cause a much more rapid ulceration, under experimental conditions, than one of normal acidity.

Lansdown and Williamson[3] have demonstrated that lymph follicles are closely associated with the pyloric type of glands, and that these occupy chiefly an area mapped out by an annular band 2 to 2½ inches to the gastric side of the pylorus, and about 1 inch distal to it, and on the gastric side this band is continued up along the lesser curvature some 2 inches towards the cardiac end over a strip of about 1 inch in width. This is, in fact, the chief ulcer-bearing area according to surgical experience. At least 75 per cent of gastric ulcers occur along the lesser curvature, and 80 per cent in the pyloric half of the stomach.

All recent theories of causation show clearly that gastric ulcer is a secondary lesion; that is, due to infection manufactured at some other part. It is not easy to prove where the factory is in all cases; but Rosenow's experiments and clinical observations suggest that the appendix is responsible in some cases at least. Of 260 cases of gastric and duodenal ulcers in which the appendix was removed, it was definitely stated to be chronically inflamed in 210; many of these were verified microscopically. That is to say, in 80 per cent the appendix was a possible source of infection. Whether the appendix is really the source, or whether the appendicular condition and the gastric lesion are both the result of a common cause, has not been finally settled. But that a chronically inflamed appendix has a marked effect on the stomach is shown in cases of appendix dyspepsia. The symptoms resemble those displayed by gastric ulcer, and at operation the pyloric half of the stomach is found to be much redder than normal, easily thrown into spasm, and there are always enlarged lymphatic glands along both curvatures; these are particularly large and noticeable along the greater curvature. Test meals show hyperacidity in a large percentage of these cases. In properly selected cases appendicectomy brings absolute relief. It is, however, true that all these patients are not cured by appendicectomy.

There seems to be a general consensus of opinion that both acute and chronic ulcers have an identical etiology; in fact, it is probably true that every chronic ulcer was in the first instance more or less acute in onset. Whether an ulcer will heal, spread, or settle down into a chronic state depends upon factors with which we are not yet familiar. Bolton's work, however, seems to show that pyloric stenosis, represented by his experimental ligature of the pylorus, is one means of making an ulcer extend. It seems to me that those cases associated with hyperacidity and secondary pylorospasm are in a rather similar condition.

The sequence of events may be as follows: First of all there is a chronically inflamed appendix; this is brought about by the double action of those two malefactors, obstruction and infection, which, working hand in hand, produce most of the lesions to which the abdominal organs are liable. Following on the chronic appendicitis, so produced, there

comes a secondary hyperacidity of the gastric juice. If the organisms in the appendicular wall are of a certain virulence, necrosis of the gastric tissues may occur as a result of infection carried by the blood-stream attacking them in the rear, and the hyperacid juice promptly digests this damaged area. The commonest situation for an ulcer is the lesser curvature, and this is, I believe, due to infection of the lymphoid tissue, which is thickly strewn here. The result is an acute ulcer. Prompt and efficient medical treatment — rest and alkalies—will usually promote healing. But if the causes which determined the formation of the ulcer persist, the ulcer will extend or the attacks will recur.

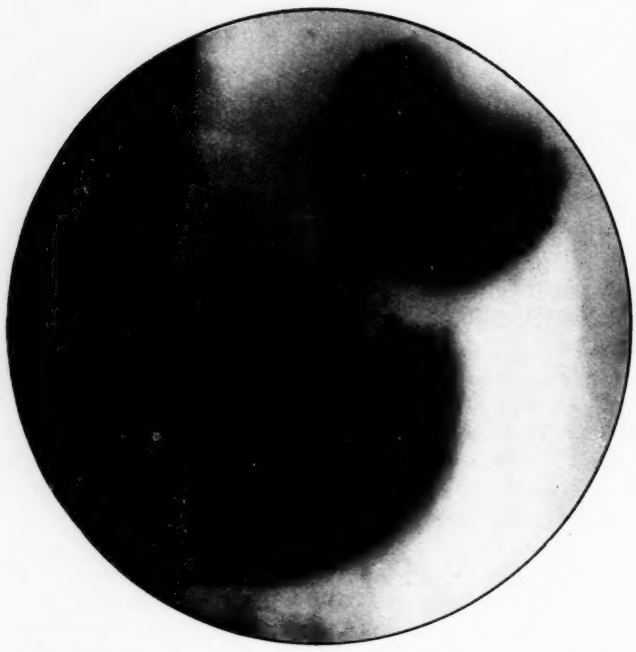

FIG 366.—Ulcer crater on lesser curvature. Large incisura. The ulcer was adherent to the left lobe of the liver.

Even if an ulcer heals under medical treatment in the first attack, it is more than probable that sooner or later the gastric juice will again digest the scar. I have already said that I believe all chronic ulcers are at first acute, and I am not at all sure that it is not true to say that most acute ulcers ultimately become chronic. It is the reappearance again and again in definite attacks which justifies the diagnosis af chronic gastric ulcer. As the attacks recur, the ulcer becomes more definitely chronic in appearance: it displays a crater of varying depth with a necrotic base, and is surrounded by hard, and at times almost cartilaginous, edges. There is usually a considerable degree of inflammatory œdema all round, and the actual cavity measures across its diameter somewhere between a threepenny and a sixpenny piece. Occasionally the crater is large enough to take the tips of all the fingers of one hand, and may still be simple, but W. J. Mayo says it is a good working rule to regard ulcers with a crater larger than a sixpenny piece as malignant.

FIG. 367.—Specimen from case radiographed in *Fig.* 366.

These chronic, thick-edged ulcers never heal. It is true the edges may, unless prevented by adhesions, become more or less approximated by contraction of the fibrous base, and may fill in with granulation tissue, which subsequently develops into fibrous tissue; but the mucous membrane is unable to grow over the base, which is in a necrotic state. Such an ulcer is not healed; and some time or other the fibrous tissue will become

digested by the gastric juice, under conditions which are not understood. It is only normal gastric mucous membrane that can withstand this digestive power. There is no regeneration of muscular fibres, and the whole thickness of the scar consists of fibrous tissue. Ulcers in this condition may remain quiescent for months or years, or they may extend in depth and perforate into the abdominal cavity, or into some neighbouring organ or structure—such as the pancreas, liver, or diaphragm; or again, they may become malignant. The contracting scar tissue deforms the organ in various ways, causing pyloric or cardiac stenosis, or hour-glass stomach; or the shortening lesser curvature approximates the two openings. Such deformities may occur although the ulcer is unhealed.

Symptoms.—Chronic gastric ulcer reveals itself by a series of attacks, separated by intervals of total or comparative freedom from symptoms. These latent periods may last a few weeks up to many months or even years, even though the ulcer is not healed, and most foods may be taken during this time with relish and impunity. Patients may, however, get reminders in these intervals that the ulcer is merely quiescent. Eventually, with absolute certainty, an attack reappears, often quite suddenly, and sometimes preceded by a state of great mental irritability. The actuating cause may be a dietetic indiscretion, a chill, over-work, mental worry, etc., probably, in some cases at any rate, owing to the production of a hyperacid gastric juice.

The average duration of symptoms in those patients who come to operation is between five and ten years. Each attack lasts about three or four weeks, and displays the same sequence of events in any one individual, vaguely spoken of by the patient as indigestion, but in reality characterized by a very definite cycle. I emphasize 'in any one individual,' because gastric ulcer in one patient may have quite a different history from that presented by another—depending probably on the position of the ulcer. But for one particular patient there is always strict repetition and regularity in the several attacks.

Pain.—There is always pain at some stage of digestion, usually one to two hours after food, relieved by more food for the same period, and also by taking alkalies, and especially by vomiting. The pain is felt in the epigastrium, and also somewhat to the left of this, and is described as burning, dull, heavy, gnawing, flatulent, or definite pain of a colicky nature. Flatulence and heartburn with acid eructations are very common. This is the usual type of attack, and may be considered as *the* type case of gastric ulcer. In another type food induces pain, the ulcer being then near the cardia, and causing cardiospasm. Curiously enough, however, an ulcer near the pylorus, especially if beginning to obstruct, may cause pain almost immediately after food. In yet a third type pain comes on two, three, or more hours after food, when it becomes practically impossible to differentiate, by symptoms alone, between gastric and duodenal ulcers. The important point is that each is a type to itself, is reproduced exactly in every attack, and disappears for varying periods, during which the patient feels quite well.

It is this regularity and periodicity which distinguishes ulcer from a multitude of conditions which otherwise closely resemble it. Such conditions are particularly dyspepsias due to appendicitis, gall-stones, cirrhosis, abdominal tuberculosis in the mesenteric glands and lower ileum, etc. This prominence of similar stomach symptoms in such apparently diverse conditions is probably accounted for by a protective spasm of the pylorus; and when the close embryological relationship of these organs is remembered, the motherly interest displayed by the stomach is not, perhaps, very surprising. If a careful anamnesis be taken, frequently it will be discovered that there have been quite definite symptoms of a chronic, grumbling appendix for years before. This is at least suggestive of cause and effect.

As time goes on, the attacks of gastric ulcer tend to become more severe, more frequent, and the character of the pain may alter. This indicates the onset of some complication. For example, severe pain in the back, usually to the left, denotes erosion of the pancreas: this occurs in some 20 per cent of long-standing ulcers. Pain which persists some time beyond the normal duration of an attack, means an imminent perforation or the supervention of carcinoma. Pain which was at first one to two hours after

food, and in the later attacks almost directly after meals, indicates pyloric stenosis or an hour-glass contraction.

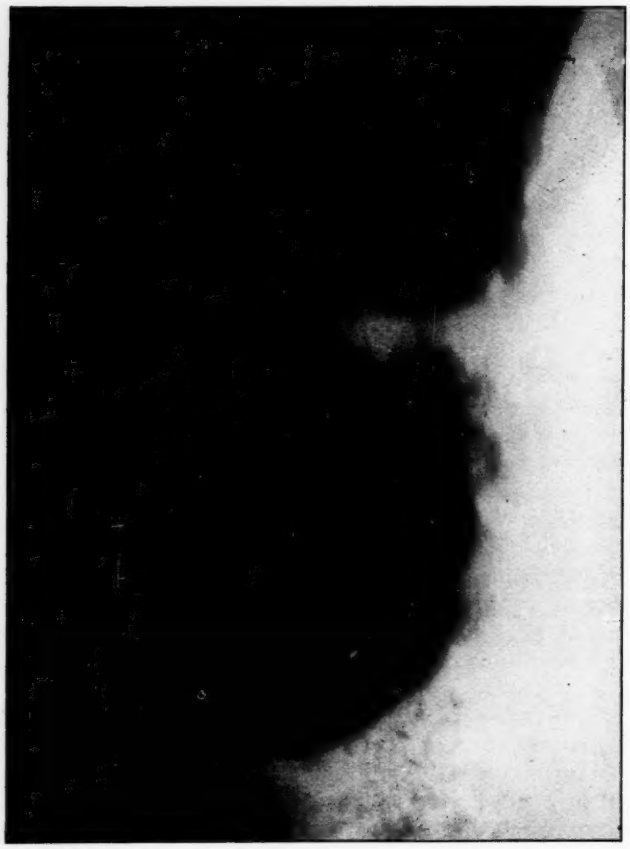

FIG. 368.—Large ulcer crater on lesser curvature, penetrating into the lesser omentum. Well-marked incisura.

As regards perforation, though it is true some acute ulcers perforate in the first few days of the initial attack, over 90 per cent of acute perforations occur during the acute exacerbations of a chronic ulcer, and so this complication is a preventable one in many cases. Yet of sixty-two gastric ulcers on which I have operated during the last two years, ten were admitted for acute perforation.

The cause of pain in gastric ulcer is probably due, not so much to food passing over the ulcer, as to spasm induced by the irritation of the ulcer by acid. The incisura which is invariably found opposite an active ulcer after a barium meal shows clearly that there is a very severe degree of spasm at this point. Hence the pain at the height of digestion, and the later onset of pain in duodenal ulcer, which is due to a similar cause. Probably pain in muscular organs, when the lesion is strictly limited to the organ, is always due to spasm and disordered motility; and this accounts for the relief brought by vomiting, as this act induces a marked degree of general muscular relaxation. Pain may arise from tethering adhesions, either because of the pull exerted on the sensitive parietal peritoneum, or again, from interference with normal motility.

FIG 369.—Specimen from case radiographed in *Fig.* 368.

Other less important symptoms of gastric ulcer are vomiting and hæmorrhage.

Vomiting is present in over half the cases. It is not a symptom of gastric ulceration unless it occurs at fixed intervals after meals, is definitely caused by the pain, and is promptly followed by relief. Patients not infrequently develop the habit of inducing

vomiting, because of the instant relief which results. The vomiting can be lessened or prevented by a liquid diet, or absolute rest; vomiting which cannot be so controlled is not due to a gastric ulcer.

Hæmorrhage, as hæmatemesis, occurs in under 40 per cent of all cases. It usually shows itself in moderate and repeated amounts, and is often associated with a very severe attack. Frequently the symptoms subside rapidly afterwards. Fatal bleeding is rare; when it does occur it is due to erosion into a large artery, such as the gastric. Hæmorrhage as obvious melæna is rather more frequent than hæmatemesis. The profuse hæmatemesis in anæmic girls is due to causes other than gastric ulceration. There seems to be great difficulty amongst some people to believe that hæmatemesis can occur apart from gastric ulcer in these girls. But surgeons, who see the state of the stomach in the living, can, by mere inspection, frequently say there is no ulcer in the organ, but that there will be found a chronically inflamed appendix. The profound blush over the pyloric end of the stomach is almost pathognomonic of this appendicular state. There is no more difficulty in explaining a hæmatemesis arising from this highly injected pyloric compartment than there is in explaining epistaxis—which not infrequently, in many people, occurs during the days when a common cold is subsiding—or hæmorrhage from any other acutely hyperæmic mucosa.

Physical Signs.—Except in the presence of complications, these are of little help in diagnosis. Epigastric tenderness is common to many abdominal conditions, and muscular rigidity only arises when perforation is impending or has actually taken place. Hyperalgesic skin areas can be elicited at times, but they are too inconstant to be reliable.

It is rare that a tumour can be felt in a case of simple ulcer; the mere presence of a palpable tumour is strongly suggestive of cancer. However, in the region of the pylorus even a simple ulcer, if accompanied by much induration, may give rise to a distinctly palpable indefinite mass, which will be tender. Also an ulcer on the anterior wall of the stomach may, when adherent to the abdominal parietes, be felt as a tender indefinite tumour.

Test meals give very varied results as to acidity, blood, etc., and the small amount of assistance obtained does not justify the extreme discomfort to which the patient has to be subjected. Some 60 per cent of gastric ulcer cases are accompanied by hyperacidity, and 40 per cent by hypo-acidity.

Having said all this about the symptoms and signs, it is nevertheless true that a clinical diagnosis of gastric ulcer is wrong in nearly two cases out of three. It is computed that 60 per cent of gastric disturbances owe their origin to a source outside the stomach, and Sir Berkeley Moynihan's epigram, that the commonest site of a gastric ulcer is in the right iliac fossa, should be constantly in the mind of everyone who thinks rashly that he can diagnose one in the stomach. There are only two methods of arriving at a certain diagnosis during life: one is by the *x* rays, concerning which Dr. Scargill has written the notes which follow; the other is by operation. But as in competent hands *x* rays give conclusive evidence in a very high proportion of cases, operation for diagnostic purposes is not often necessary.

Treatment.—Until more is known of the actual course of *acute* ulcers, medical treatment is the proper means to adopt. But there is a surgical aspect even at this stage, for three reasons:—

1. The ulcer is a secondary manifestation of some distant infection, frequently, I believe, in the appendix; and when the acute attack is over, the primary source should be sought for and treated.

2. There is no doubt that an acute ulceration is a potential source of acute peritonitis by perforation; several cases have been reported.

3. An acute attack does not always mean an acute ulcer. The initial attack probably does, but the recurrent attacks of a chronic ulcer are frequently quite acute.

The treatment of *chronic* gastric ulcer and its complications is always surgical. There cannot be any hesitation in accepting this. There is not unanimity, however, as to the operation that should be performed.

Such operations as gastroplasty and the like are defunct; but there still remain quite a multitude, viz., gastro-enterostomy (anterior and posterior), excision, trans-gastric excision, excision and gastro-enterostomy, gastrogastrostomy, Balfour's operation, jejunostomy, and partial gastrectomy. The mere number shows that the results are not all that could be desired. This is in marked contrast to the one and only operation for duodenal ulcer.

I think, however, when all the facts are considered, it must be conceded that there ought to be only one operation for the majority of gastric ulcers, viz., partial gastrectomy. Excision alone has been almost finally abandoned; if excision is to be done at all it should be combined with gastro-enterostomy. Most surgeons merely do a gastro-enterostomy and infold the ulcer. Any method of excision is, in my opinion, more difficult than gastrectomy.

GASTRECTOMY.

For the following reasons gastrectomy should be the operation of choice :—

1. In quite a large percentage of cases of gastro-enterostomy there is a persistence of the ulcer. I have been unable to get figures for the Leeds General Infirmary showing the after-results of the operation. But at the Middlesex Hospital,[4] of 65 cases of gastric ulcer treated by gastro-enterostomy, 46 per cent were cured, 20 per cent relieved, 11 per cent slightly benefited, and 23 per cent total failures. Of 67 cases treated at St. Mary's Hospital,[4] 42·2 per cent were cured and 38 per cent gave bad results. The cases in which gastro-enterostomy can be relied on to do much good are those where the ulcer is situated close to the pylorus, being associated with stenosis. But even here the ulcer may subsequently give trouble.

As regards the after-results of gastrectomy, it is not possible to speak with great assurance, owing to the comparatively small number of cases that have been treated by this method. During the first five of the seven years reviewed for the purposes of this paper, there were only 13 cases of gastric ulcer treated by gastrectomy at the Leeds General Infirmary. During the last two years I have myself done over 70 gastrectomies, 44 of them for gastric ulcer. I have written to about half the number, or their doctors, and 16 have replied. This is not a large number from which to draw conclusions, but, taken for what they are worth, the replies are distinctly encouraging. Of these 16 cases, one says he is no better, though he admits he has not such severe indigestion as before; 3 feel uncomfortable after food, but have no actual indigestion. The other 12 are quite well; they all say they have never been better in their lives, can eat anything, can take normal-sized meals, and have no discomfort afterwards. They are all back at work, one a miner working at the coal face.

Gastrectomy cases probably require more careful feeding for a considerably longer time after operation than gastro-enterostomy patients. The loss of a considerable part of the stomach must be a condition requiring time for readjustment of function. Probably the discomfort some of the patients have had is due to eating freely and the food going rapidly into the jejunum and causing an unwonted distention there. Sir Berkeley Moynihan, who is the only other surgeon in the British Isles who has regularly practised this operation for gastric ulcer over some three years, has a larger series of cases, and his results are excellent.

2. The secondary advantage of gastrectomy over any other operation has reference to carcinoma of the stomach. Carcinoma in other situations so commonly follows prolonged irritation—for example, in the tongue, breast, and indeed in most parts where it occurs—that it is probable, on the face of it, that cancer should arise at times in such chronic ulcers as those in the stomach. But there is more convincing evidence than mere probability. At least 6 per cent of the ulcers of my series have proved, on microscopic investigation, to be early carcinoma. This percentage almost certainly under-estimates the frequency of this change, because all the stomachs were not examined. One case was very interesting in this respect. The man had had attacks of gastric ulcer for five years; the pain in the last attack had persisted for three months instead of disappearing in as

many weeks, as had previously happened. At operation it was thought the ulcer was simple, and indeed all the characteristics of a simple ulcer were presented on inspection of the specimen afterwards; on microscopical examination, however, it was pronounced a definite early carcinoma.

According to Kocher, 5 per cent of gastric ulcers treated by gastro-enterostomy become malignant sufficiently long afterwards to make it improbable that the ulcer was malignant in the first instance. Taking the cases of well-established carcinoma of the stomach, about 60 per cent are computed to have originally been simple ulcers: Mayo says 65 per cent, Moynihan 60 per cent, Wilson and McCarty 71 per cent, Sherren 40 per cent.

This is a very strong argument for gastrectomy as the routine operation for gastric ulcer, and also for the treatment to be undertaken at a much earlier date in the history of the disease than surgeons are in the habit of seeing them at present.

3. The third point for gastrectomy relates to the other complications: hæmorrhage, perforation, and stenosis. I distinctly remember a case at the Leeds General Infirmary which died after gastro-enterostomy as a result of profuse hæmorrhage, and at the post-mortem the open end of a vessel was found in the base of the ulcer. Several others have been reported.

As regards stenosis, in the form of hour-glass stomach, the usual operation has been gastrogastrostomy. According to Walton, 30 per cent of these cases after this operation receive no benefit or relapse. It is, perhaps, not surprising, when it is remembered that there is usually still an active ulcer present.

Perforation of the ulcer is another complication which is known to occur after gastro-enterostomy. Sir Berkeley Moynihan has operated on such a case recently. The patient was a woman who had an acute perforation of a gastric ulcer; this was sutured, and she recovered. Within a few months another acute perforation occurred of another ulcer; this was sutured, and a posterior gastro-enterostomy was performed. After this another acute perforation took place of a fresh ulcer; this was sutured, and she eventually developed a jejunal ulcer. Perforation is very dangerous, and only occurs in a small percentage after operation, but it is not right that the risk should be run when it can be altogether prevented. Taking all cases of gastric and duodenal ulcers, before operation, there is about an equal liability to acute perforation; but the death-rate is much higher in the case of a perforated gastric ulcer. Amongst my own cases, the death-rate in duodenal perforation is 24 per cent, in gastric it is over 40 per cent. This is due to the greater amount of extravasated material from the stomach, and to the higher septicity of the stomach contents causing a more rapid and severe general peritonitis.

It cannot be said that the best is being done for one's patients if an operation is chosen which leaves so much to chance as gastro-enterostomy has been shown to do, when there is another method of operation which is free from these risks, unless this alternative method has greater shortcomings, or as great. The only serious objection that can be made against gastrectomy that I know of is the slightly higher operative mortality. The difference is very small. During the seven years under review, the operative mortality of gastro-enterostomy for gastric ulcer was 5 per cent. To this must be added that due to the onset of carcinoma and the other complications after an apparently successful operation. This number is difficult to estimate, but it probably more than makes up the slight difference, for the operative mortality of gastrectomy is only 7 per cent. In addition to this, the many failures after gastro-enterostomy must be weighed against the results of gastrectomy, which up to the present indicate a much higher percentage of absolute cures. Gastrectomy is undoubtedly a more serious operation in some cases, but in the majority, though it takes rather longer to do, it is not any more difficult. Four-fifths of all gastric ulcers begin in the pyloric half of the stomach, and are, therefore, easily removable. It is the unusual position near the cardiac end which creates the extra risk, and it is precisely in these cases that gastro-enterostomy is most likely to be followed by a poor result.

The Operation of gastrectomy consists in removing as much of the stomach as is necessary to get about 1 inch of healthy mucosa beyond the ulcer on the cardiac side, and

anastomosing the side of the jejunum to the cut end of the stomach. The resection is begun by dividing the duodenum just beyond the pylorus, and freeing the stomach to beyond the ulcer. Doing the operation this way allows of the stomach being used as a retractor when the more inaccessible cardiac end is being dealt with. If the pancreas be adherent, as it is in 20 per cent of cases, a piece of it should be cut away, leaving it attached to the ulcer. This makes no difference to the mortality, but it is surprising how easy an apparently difficult case becomes after dividing this tethering adhesion. It is essential to investigate the stomach for more than one ulcer, for though it is unusual to find more than one, yet it does occur in a small percentage. The appendix should be removed as a routine, provided the patient is able to stand the extra manipulation. This organ has possibly something to do with the causation of the ulcer, and in any case it is better away. Gall-stones are present in 1·5 per cent of gastric-ulcer cases ; and duodenal ulcers are associated with gastric in 2·5 to 3 per cent. The former should be dealt with as circumstances permit ; the latter can easily be included in the resected area by placing the clamp on the duodenum a little more to the right.

There is one point of practical importance in the differentiation of gastric and duodenal ulcers. Ulcers well on the body of the stomach are easily recognized as gastric, but near the pylorus confusion may arise. Indeed, some surgeons, especially the Swiss, say it is impossible to be sure on which side of the pylorus the ulcer is. There is no occasion for this confusion, except in those uncommon cases when the ulcer is actually at the pylorus. The exact position of the pylorus is marked on the surface of the stomach by a thin white line ; just to the gastric side of this is an unmistakable branching vein, which ascends from the lower margin of the pyloric region. These two landmarks do not coincide exactly, but for practical purposes they may be considered to do so. The white line cannot always be seen ; the vein invariably can. Any ulcer to the left of this is gastric, and of course one to the right is in the duodenum. It is of importance to differentiate the site of the ulcer, because the treatment of the two conditions should be quite different.

There is one position in which gastro-enterostomy has the advantage over gastrectomy, and that is when the ulcer is high up on the lesser curvature. In some of these cases it is impossible to get sufficiently beyond the ulcer for resection, especially when there is much surrounding induration, and then the operation devised by Sir Berkeley Moynihan is an excellent substitute. This consists in doing an anterior gastro-enterostomy en Y, and stitching a catheter into the jejunum as it comes from the duodenojejunal flexure to the entero-anastomosis. This gives absolute rest to the stomach for as long a period as may be desired. It may be used, too, for ulcers which by their size are unsuited to gastrectomy. Sir Berkeley Moynihan has reported one such case ; the patient had neither a bite nor drink by mouth for two and a half years. The subsequent closure of the jejunostomy only narrows that part of the intestine, which acts to all intents and purposes merely as a common bile-duct. The opening in the stomach is made so high up that, if an hour-glass contraction develops as a result of healing of the ulcer, exit from the proximal compartment is adequately provided for.

It may be said that gastrectomy removes an unnecessarily large amount of the stomach, but I think I have shown clearly that the part removed is the chief ulcer-bearing area ; that once an ulcer has formed, the possibility of carcinoma being grafted on to it is a very real one ; that so far as we know at present no functional disability results ; that the return to health is practically as quick as, and certainly more sure than, after any other operation ; and that with a proper technique, such as has now been evolved, the operation is no more difficult than any of the other usual methods of treatment.

THE X-RAY EXAMINATION FOR GASTRIC ULCER.

It is ten years since Reich, a German radiologist, first demonstrated the presence of a gastric ulcer by means of a bismuth meal, and his diagnosis was proved at operation. For some years following this, the results of bismuth-meal examinations were uncertain

and unreliable; each observer had his own method of observation and technique, and interpreted the various signs differently.

During the last four or five years great progress has been made. Apparatus has been improved, technique has been developed by the experience gained, and, most important of all, the various *x*-ray signs have been analyzed and sifted, the information obtained has been orderly arranged, and the whole subject put on a proper foundation.

For these advances in our knowledge we are greatly indebted to some of the American radiologists, and especially to Carman, of the Mayo clinic. A keen and original observer, a lucid writer, with a great amount of material to work on, he has caused the whole radiological world to follow his technique and teaching. His results were at first received by many with scepticism. To-day the diagnosis of gastric ulcer by *x*-ray examination is far more certain than the clinical diagnosis. Nine-tenths of all ulcers give definite *x*-ray signs (*Figs.* 366, 368); and in the great majority of cases the signs are pathognomonic.

The Method of Examination now almost universally employed is that originally introduced by Haudek in 1912. The day previous to examination the bowels are thoroughly opened, and light diet only is taken. On the day of examination, six hours before the patient comes to the *x*-ray room, a bismuth or barium meal is given, on an empty stomach. The stomach must be empty: the importance of this is not fully realized; food residue may cause deformity of stomach contour simulating deformity due to disease. The meal given must be of thin consistency; a thick stodgy meal, of porridge or bread and milk, is useless for the diagnosis of gastric ulcer.

At the time of examination the position of the six-hour meal is located, any residue in the stomach being noted: a normal stomach should empty itself of the amount given (16 fluid ounces) in three to four hours. There is considerable latitude in the emptying-time of a normal stomach; any opaque material left in the stomach at the end of six hours is definitely abnormal. The great advantage of the six-hour-meal method of examination is that it obviates the repeated examinations that are made by some radiologists to determine exactly when the stomach empties. It is impossible to do this in hospital practice. If there is no opaque material present after six hours, the motility of the stomach may be considered normal. The second meal is given—sufficient to fill the stomach completely—and the examination proper of the stomach is made.

X-ray Signs of Gastric Ulcer.—(1) The direct or primary sign—the demonstration of the actual ulcer cavity. (2) The indirect or secondary sign—the spasm caused by the ulcer.

The Direct Sign.—A small erosion of the mucous membrane only will not show a projection from the lumen of the stomach and cannot be demonstrated. If the ulceration has penetrated into the wall of the stomach, or further, into the lesser omentum, and is possibly adherent to, or has penetrated, the liver or pancreas, the cavity formed can be demonstrated filled with the opaque material.

The great majority of ulcers occur on the lesser curvature, or on the posterior surface close to the lesser curvature; these can be seen either in anteroposterior or semilateral view. A small percentage of ulcers occur on the posterior surface. These can usually be seen when the stomach is emptying. Ulcers occurring on the anterior or posterior surface close to the pylorus cannot easily be demonstrated.

The Indirect Sign.—The incisura or spasmodic hour-glass. In a majority of gastric ulcers there is to be seen a remarkable spasmodic contraction of the circular fibres of the stomach opposite, or very nearly opposite, the ulcer, producing an indentation of the greater curvature of varying degree. The contraction may be so strong as to produce an apparent bisection of the stomach—a spasmodic hour-glass. These incisuræ occur also in the stomach reflexly from extrinsic causes, chiefly in conjunction with disease of the appendix, lower ileum, gall-bladder, kidney, and duodenum. The intrinsic incisura may persist for a few seconds, or until the stomach is nearly empty, almost always the latter. If due to reflex causes outside the stomach, the incisura can be made to disappear by injecting atropine hypodermically. This is not practicable as a routine method, as doses have to be given sufficient to produce physiological effect before the inhibiting effect on

the stomach is produced, and severe poisoning may occur. It is only safe to use this test if one is quite certain that there is no personal idiosyncrasy to atropine.

The presence of an incisura may be taken as strongly presumptive of gastric disease. If the stomach is examined when too full and distended, the incisura may be obliterated by the upper and lower portions of the stomach coming in contact with each other. Palpation along the greater curvature will sometimes demonstrate it when not at first visible.

An ulcer in the pyloric portion of the stomach will sometimes cause a spasmodic contraction of the greater curvature so strong, and to such a depth, that it causes a kind of pseudo-pylorus. This is an important point to bear in mind, for it strongly simulates pyloric stenosis due to duodenal ulcer.

If an incisura is seen on the greater curvature, and on the lesser curvature a projection of varying size is seen, an absolutely definite diagnosis of gastric ulcer can be made. The whole diagnosis is made by the fluoroscopic-screen examination; a radiograph records only what is seen at one stage of the examination.

With efficient apparatus, careful attention to modern technique, and correct interpretation of the signs given, the *x*-ray examination is the most valuable means of diagnosing the presence of an ulcer, short of laparotomy.

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[3] Lansdown and Williamson, *Brit. Jour. Surg.* Vol. ii, 306.
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FRACTURE OF THE ATLAS VERTEBRA.

REPORT OF FOUR CASES, AND A REVIEW OF THOSE PREVIOUSLY RECORDED.

By GEOFFREY JEFFERSON, Manchester.

Fractures of the atlas vertebra form one of those categories of rare accidents possessed of a considerable interest. This is particularly true of the cases in which the injury has not been attended by fatal results. My attention having been drawn to the subject by two recent cases, I turned to the literature, and found that so few cases had been recorded, and these in such diverse places, that it seemed well they should be collected together under one head, so that a clearer picture might be obtained of the types of fracture, its concomitant symptoms, and the results which are to be expected from it. My first impression was that fracture of the atlas would be almost invariably followed by death. Greater

Fig. 370.—Fracture of the posterior arch of the atlas vertebra in two places. Second cervical vertebra intact.

knowledge of the subject has, however, modified this opinion. No doubt, if the atlas was morphologically similar to the other vertebræ, death would be the common result of fracture. But the atlas presents so many peculiarities of shape, function, and relationship, that the accidents which may happen to it present many differences from those to which the other vertebræ are heir. The most interesting aspects of the question are the genesis of atlas fracture, the mechanism by which it comes about, and the displacement which follows. I have attempted below to explain these, and to give a new and more physiologically accurate account of the manner in which injuries, and more particularly fractures, of this vertebra are brought about.

I am able to report one clinical case of fracture of the posterior arch, one of fracture

of the left lateral mass by a gunshot injury, and two pathological specimens hitherto undescribed—one from the Museum of the Royal College of Surgeons, the other from the Pathological Museum of Manchester University.

Case 1.—Lieut. B., R.A.F., dived his machine into a bank of telegraph wires on a misty morning, Jan. 1, 1919. He struck the wires on the level, travelling at about 120 miles per hour. He remembers seeing pieces of propeller, wing, and strut fly past him, and then nothing more. It appears that he was thrown out of the plane, and fell some distance on to his head. He recovered consciousness twelve hours later in a C.C.S. and says "he felt very fit," except that his neck was stiff and sore. No neurological signs were detected; no headache.

He was admitted to No. 14 General Hospital two days later. His general condition was so good, and he made so light of his symptoms, that no serious injury was suspected. The stiffness of the neck persisting, he was radiographed, and I was asked to see him by Captains Gardiner-Hill and D. H. Fraser. The patient complained of deep-seated pain in the upper part of the neck, and inability to nod his head. The most painful movement was extension of the head, no doubt because this movement tends to compress the arch of the atlas between the occiput and the axis. Flexion of the head was possible through a few degrees only, and rotation was quite painless, so that he could look over his shoulder. All movements needed time, and were executed slowly. He raised himself in bed with a very characteristic action, placing his left hand under the suboccipital region as a support, and then, catching hold of the side of the bed-frame with his right hand, he pulled himself up into the sitting posture. On examination there was no swelling of the neck, but there was distinct tenderness on deep pressure in the suboccipital triangles behind. Pressure on the anterior arch of the atlas through the mouth produced no pain, nor was anything abnormal to be felt there. There were no signs of cord injury, a neurological examination being negative; no difficulty in swallowing, and neither neuralgia nor anæsthesia in the distribution of the great occipital nerve. Captain Dale's radiograph (*Fig.* 370) shows the posterior arch of the atlas fractured in two places at the site of the groove for the suboccipital nerve and occipital artery. The fragments are not displaced, being held in excellent position by the ligaments and periosteum. This lack of displacement, no doubt, accounts for the absence of cord or nerve injury. An antero-posterior view through the mouth revealed an intact odontoid process. The fracture is, therefore, an 'isolated' atlas fracture.

A Lorenz plaster bed was made for the patient, and he was transferred to England. A later *x*-ray photograph showed positive signs of new bone formation at the sites of the fracture of the neural arch. He remained in the Lorenz bed for two months; convalescence was smooth.

I saw him again three months later, and he had practically quite recovered. The head was held a little stiffly as he walked, but movements were free and painless.

Case 2.—Pensr. J. C., age 22. Gunshot wound of neck, Oct. 4, 1917. Entrance over the angle of left mandible, exit just below inion. From the position of the wounds it is evident that the track of the bullet involves the left lateral mass of the atlas. The head is held very stiffly; true power of movement in the occipito-atlantal joint is almost nil. He nods stiffly with his neck, flexing and extending through the lower cervical joints. The same is true of lateral movement: His main trouble is occipital neuralgia, which is severe. The scalp is hypersensitive in this area, and he cannot brush his hair. A radiogram shows an indefinite irregularity in the shadow of the left lateral mass. It is difficult to make out whether the occipito-atlantal joint surfaces are affected.

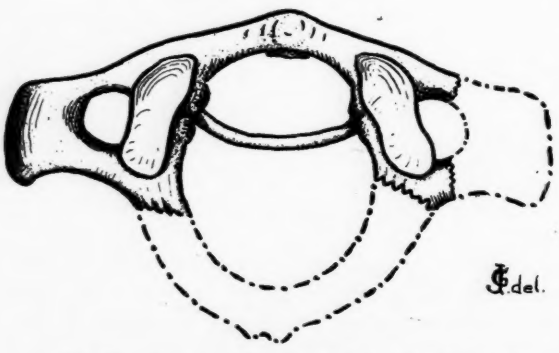

FIG. 371.—Fracture of the posterior arch of the atlas vertebra in two places, with fracture of the right transverse process. Fractured parts missing. Royal College of Surgeons Museum, Catalogue No. 2021.

Case 3.—*Museum Specimen No.* 2021, *Royal College of Surgeons. Series, Diseases of the Vertebral Column.*

This dry specimen (*Fig.* 371), originally in the private collection of Mr. Langstaff, consists of the upper three cervical vertebræ of a man thrown from his horse and instantaneously killed. The posterior arch of the atlas was broken off and is missing. The fracture runs through the groove for the suboccipital nerve and vertebral artery on each side. The right transverse process is also broken off and missing, the fracture passing through the foramen for the vertebral artery. The odontoid process and axis are intact; the transverse ligament is unruptured. The notes in the catalogue state that the spinal cord and dura mater were lacerated, but beyond this there is no description.

Case 4.—*Manchester University Pathological Museum. Specimen No.* 738.

This dry specimen consists of the articulated atlas and axis vertebræ (*Fig.* 372). The atlas ring is broken at two points, the anterior arch near its junction with the left lateral mass, the posterior arch on the left side also. The atlas is a strong one and the posterior arch firmly moulded. The atlas fracture is complicated by a break of the odontoid, the upper half of this process being wanting. No clinical history is attached to this specimen, but that the patient survived the accident for a time is evidenced by a slight bone-formative process in front.

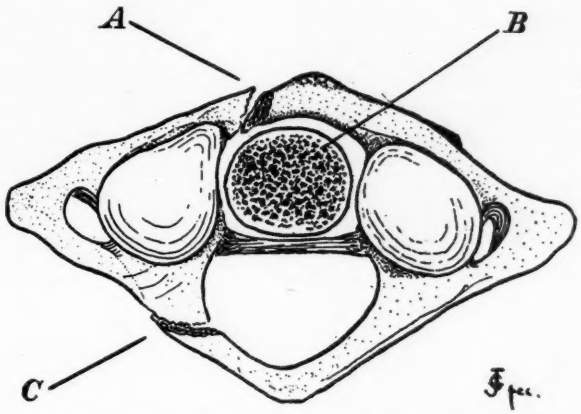

FIG. 372.—Fracture of the anterior (A) and posterior arch (C) of the atlas vertebra. Fracture of odontoid process (B) of axis. Manchester University Pathological Museum, Catalogue No. 738.

Cases 1 and 3 are examples of a precisely similar injury, the 'isolated atlas fracture' of German writers. In *Case* 4 the fracture of the atlas is complicated by an injury to the odontoid. Of the four cases, only *Case* 3 ended in sudden death, from laceration of the lower end of the medulla and cord; *Case* 4 appears to have survived the injury for a time, whilst *Cases* 1 and 2 recovered completely.

From so small a number of cases it is impossible to draw any valuable deductions as to the mode of fracture and its usual result. The injury is not a common one, though not so uncommon as might be thought. I have been at considerable pains to collect the cases in which fracture of the atlas has been described by previous writers. I found 18 cases of isolated atlas fracture, and 24 in which another vertebra had suffered hurt at the same time, 46 cases in all, counting the 4 just described. In the tables appended will be found the salient points of the known examples; the cases are arranged in the alphabetical order of the reporters' names. As the circumstances in which the injury has been brought about are important for a consideration of the mechanism of the fracture, I have indicated the nature of the accident in each case. A bibliography will be found at the end of the paper.

Table I.—**ISOLATED FRACTURES OF ATLAS—21 CASES.**

AUTHOR	NATURE OF ACCIDENT	CLINICAL SIGNS OF CORD OR NERVE INJURY	ANATOMICAL DIAGNOSIS	RESULT
No. 1. Betz	Fell on neck and shoulder	Progressive paralysis of arms and legs	Isolated fracture of posterior arch of atlas	Died 3 months later. Half-cm. piece of bone driven against medulla. Focal hæmorrhage in medulla
No. 2. Cooper, Astley (Cline's case)	Severe fall, injuring neck (3-year-old boy)	None	Both arches of atlas broken across	Died 12 months later (intercurrent disease presumably). No mention of condition of cord at autopsy
No. 3. Corner	Rolled out of chair with head flexed	No cord signs. Severe neuralgia great occip. nerve	Fracture of atlas	Recovered
No. 4. Delorme	Gunshot wound of neck	None	Fracture posterior arch of atlas. Vertebral artery compressed by bone fragments	Died 10 days later as a result of secondary hæmorrhage. Internal carotid had been severed

Continued on next page

Table I, continued.—Isolated Fractures of Atlas.

Author	Nature of Accident	Clinical Signs of Cord or Nerve Injury	Anatomical Diagnosis	Result
No. 5. George	?	No clinical details	Fracture post. arch of atlas, two places	Presumed recovered
No. 6. George (Butler's case)	?	No clinical details	Fracture post. arch of atlas, one place	Presumed recovered
No. 7. Holding	Fell down stairs on to back of head and shoulders	No cord signs. Severe occipital neuralgia	Isolated fracture of atlas	Recovered
No. 8. Jefferson	Aeroplane crash; fall on to head	None	Fracture post. arch of atlas, two places	Recovered
No. 9. Jefferson	Gunshot wound	None. Neuralgia left great occipital nerve	Fracture left lateral mass	Recovered
No. 10. Jefferson (Langstaff's case)	Thrown from horse	Laceration brain stem	Fracture post. arch of atlas, two places	Died immediately
No. 11. Ludloff	? (See bibliographical note)	?	Fracture left lateral mass of atlas	Recovered
No. 12. Marshall, J.	Fell from second storey window	Monoplegia right arm, progressing to triplegia, both arms and right leg	Fracture right lateral mass of atlas	Died 9 days later, infection of fracture, with myelitis of cord
No. 13. Milner	Fell off roof, neck probably flexed	Medulla divided	Posterior arch of atlas fractured in two places. Complete dislocation of occiput from atlas	Died instantaneously. Medulla found divided at post-mortem
No. 14. Park	? (See bibliographical note)	?	Fracture anterior arch of atlas	Recovered. Caries of atlas, extrusion of sequestrum by the mouth 45 days later
No. 15. Quercioli	Fell from tree	No limb palsies. Dysphagia	Atlas broken into four pieces by symmetrical fractures of both arches	Died 13 days later from pneumonia ab ingestis. Cord found uninjured at autopsy
No. 16. Schneider	Fell down stairs	Monoplegia right arm; recovered	Fracture anterior arch of atlas	Recovered.
No. 17. Smith	Shot through mouth (suicide)	Monoplegia right arm	Fracture anterior arch of atlas	Died suddenly 6 weeks later, myelitis of brain stem
No. 18. Stokes	Shot in neck	Clinical signs of spinal meningitis	Fracture right upper articular facet of atlas	Died 6 days later, suppurative meningitis
No. 19. Sicard and Roger	Bale of paper dropped on to head from a height	No cord signs. Anæsthesia left great occipital nerve	Fracture posterior arch of atlas, left side	Died 1 month later of pneumonia. No injury to cord; left suboccipital nerve almost divided by bone fragment
No. 20. Sicard and Roger	Fell 6 feet on to head	No cord signs. Anæsthesia left great occip. nerve	Fracture posterior arch of atlas, left side	Recovered
No. 21. Sicard and Roger	Struck by a beam on left side of back of head and neck	No cord signs. Anæsthesia left great occipital nerve	Fracture posterior arch of atlas, left side	Recovered

Of the 21 cases in *Table I*, 7 died as a direct result of the injury (i.e., *Cases* 1, 10, 12, 13, 15, 17, 18), or 35 per cent, and 3 more from other causes (*Cases* 2, 4, 19). Only 2 were killed outright, and 11 recovered (50 per cent). In 6 the cause of death was injury or infection of the cord and meninges (*Cases* 1, 10, 12, 13, 17, 18). In Quericoli's case the head hung forward on the sternum, the patient could not swallow, and he died from inspiration pneumonia. Delorme's case died of secondary hæmorrhage from the internal carotid (a gunshot wound). There is no evidence that Astley Cooper's case, a little boy of three, died as the result of the injury. Most writers have taken it for granted that he died of intercurrent disease, but actually Cooper says nothing of the cause of death.

Of the 11 patients who recovered, all seem to have done well. It is noteworthy that only Schneider's case had a cord injury, resulting in a limb palsy, and this passed away. Six are stated to have had either neuralgia or anæsthesia of the great occipital nerve.

Table II.—**COMPLICATED FRACTURES OF ATLAS—25 CASES.**

Author	Nature of Accident	Clinical Signs of Cord or Nerve Injury	Anatomical Diagnosis	Result
No. 1. Van Assen	Fell 13 feet head foremost	No cord signs. Occipital neuralgia	Fracture both arches of atlas. ? Fracture of odontoid. ? Rotatory dislocation of atlas on axis	Recovered
No. 2. Bell, Charles	Fell 50 feet on to shoulders	——	Fracture (? posterior) arch and portion of body of atlas. Odontoid process broken off	Instantaneous death, presumably from medullary injury. Autopsy, but no mention of condition of brain stem
No. 3. Berndt	Fell forwards down steps	Diplegia right arm and leg, anæsthesia of left side (Brown-Séquard palsy)	Transverse fracture anterior arch of atlas. Odontoid broken off. Rotatory dislocation of atlas on axis	Died one month later. Compression of right half of cord, no gross lesion of brain stem
No. 4. Blackwood	Fell 4 feet on to right side of head	Total paralysis below level of larynx. Kept alive for 34 hours and 40 mins. by artificial respiration	Atlas fractured in 3 places, both arches. Odontoid broken off. Dislocation of occiput on atlas	Died 35 hours later, hæmorrhage into cord from level of foramen magnum to third cervical vertebra
No. 5. Blanc	Hit on back of neck with heavy timber, head flexed	Concussion. Diplegia both arms	Fracture of atlas. Fracture of axis. Forward luxation of atlas on axis	Recovered. Palsies improved when seen 6 years later
No. 6. Boeckel	Fell some 12 feet head foremost on to sand	None	Fracture right lateral mass of atlas. Rotatory dislocation of atlas on axis	Recovered
No. 7. Brooks	A fall. No details	Monoplegia left arm. Speech thick	Fracture of both arches of atlas in two places. Odontoid broken off	Died suddenly 24 hours later, presumably from injury to cord
No. 8. Corner	Fell off horse on to forehead	Dysphagia and thick speech. No affection of limbs	Fracture of anterior arch of atlas. Rotatory dislocation of atlas on axis. Condition of odontoid doubtful	Recovered

Continued on next page

Table II, continued.—Complicated Fractures of Atlas.

Author	Nature of Accident	Clinical Signs of Cord or Nerve Injury	Anatomical Diagnosis	Result
No. 9. Corner	Museum specimen, history unknown	—	Atlas mis-shapen by healed fractures: (1) Comminuted fracture right lateral mass; (2) Fracture posterior arch in centre. Atlas ankylosed to occiput. Fracture body of axis	Survived some time
No. 10. Hamilton	Fell 40 feet on to vertex	Paraplegia below the nipple level. Arms normal	Fractures of both arches and right transverse process of atlas. Odontoid uninjured. Fracture 6th cervical vertebra	Died 48 hours later. Cord compressed at level of 6th cervical vertebra
No. 11. Jefferson	Unknown (museum specimen)	Unknown	Fracture of anterior and posterior arches of atlas. Fracture of odontoid	Lived a short time
No. 12. Kocher	Fell 10 feet on to head	None	Probable fracture of atlas and of odontoid	Recovered
No. 13. May	Fell 15 feet on to back of head	No definite cord signs; fully recovered later	Fracture of posterior arch of atlas. Odontoid broken off. Rotatory dislocation atlas on axis. Fracture transverse processes 5th and 6th cervical vertebrae	Died 3½ years later, maniacal, suffocated with piece of meat. Groove for first cervical nerve found absorbed at autopsy. Cord normal
No. 14. McCarthy	Fell head foremost down hold	Quadriplegia arms and legs	Atlas broken into five fragments. Odontoid broken off	Died 3½ days later. Cord concussed, no compression
No. 15. Melchiori	Fell off ladder	—	Posterior arch of atlas broken in two places. Odontoid broken off. Dislocation atlas on axis	Died instantaneously. Brain stem compressed
No. 16. Mixter and Osgood	Fell down 13 stairs	Severe occipital neuralgia. No cord signs	Fracture of anterior arch of atlas. Rotatory dislocation of atlas on axis	Recovered
No. 17. Mixter and Osgood	Railway accident	All four limbs became spastic 2½ months after accident	Fracture posterior arch of atlas. Complete dislocation of atlas on axis	Died suddenly one month after laminectomy and five months after injury. No autopsy.
No. 18. Mixter and Osgood	Fell from a height on to right side of head and neck	Right occipital neuralgia. Monoplegia right arm	Fracture posterior arch of atlas, right side. Rotatory dislocation of atlas on axis	Recovered
No. 19. Phillips	Fell off hay rick on to occiput	None	Fracture posterior arch of atlas in two places. Dislocation of remaining part forwards. Odontoid process broken off	Died 47 weeks later of anasarca (? nephritis). Fracture had done well.

Table II, continued.—Complicated Fractures of Atlas.

Author	Nature of Accident	Clinical Signs of Cord or Nerve Injury	Anatomical Diagnosis	Result
No. 20. Picqué (Billot's case)	Fell 11 feet	Difficulty in swallowing, no limb palsies	Fracture of anterior arch of atlas. Rotatory dislocation atlas on axis	Recovered
No. 21. Pilcher	Fell 15 feet on to forehead	Diplegia right arm and leg, improved by laminectomy. Palsy developed 2½ months after accident. Occipital neuralgia	Fracture lateral mass of atlas, body of axis compressed. Rotatory dislocation of atlas on axis, with which it is ankylosed	Recovered. Seen 9 years later: right hand weak, leg recovered
No. 22. Scott	Hit on back of neck with heavy stick	None. Walked about	Fracture of both arches of atlas in middle. Odontoid broken off	Died 10 days later of tetanus. Cord normal
No. 23. South	Fell downstairs	Quadriplegia, with hyperæsthesia right half of body	Atlas broken in two places. Odontoid broken off. Fracture 5th cervical vertebra	Died 5 days later. Hæmatomyelia level of 5th cervical vertebra.
No. 24. Spangenbern	Fell off horse on to head	None	Fracture posterior arch of atlas. Odontoid broken off	Died 15 months later Osteomyelitis of atlas and axis. Cord normal
No. 25. Speyer	Fell on head	Monoplegia right arm, progressing to other limbs	Fracture both arches of atlas. Odontoid broken off	Died 10 days later. No gross injury to cord. Blood extravasation over medulla

Of the 25 cases in *Table II* of atlas fracture complicated with injury to other vertebræ, 10 died from cord injury (*Cases* 2, 3, 4, 7, 10, 14, 15, 17, 23, 25), 1 from tetanus (*Case* 22), 1 from nephritis (*Case* 19), and 1 was accidentally choked long after the accident (*Case* 13). The last 2 cases might be regarded as recoveries, as was Corner's second case, in which the cause of death is not known (a museum specimen). In the specimen which I have described, the bone reparation is not so extensive as to suggest that the patient long survived. Only 2 of these 25 cases were killed outright (*Cases* 2, 15). Signs of cord injury were present in 3 out of the 10 cases which recovered (*Cases* 5, 18, 21)). Blanc's case had a severe cerebral contusion which may have contributed to the palsies. Only 10 cases recovered (*Cases* 1, 5, 6, 8, 12, 13, 16, 18, 20, 21).

The bone most commonly involved in the injury in addition to the atlas was the odontoid process. The next most common injury was a subluxation of the atlas on the axis, and less commonly a fracture of a vertebra at a distance.

Table III.—Complicated Atlas Fracture.

Nature of Supplementary Injury.

		Total
Fracture of odontoid	certain	15
	doubtful	2
Fracture of axis		2
Fracture of lower cervical vertebræ	V.	1
	V. and VI.	1
	VI.	1
Dislocation of atlas on axis	certain	11
	doubtful	1

Taking the whole series of 46 cases, there were 16 deaths from cord injury, and this is commoner in the complicated than in the isolated fractures, roughly in the proportion of three to two. Signs of cord injury were lacking in 19 fractures—11 isolated and 8 complicated. This shows that fracture of the atlas does not itself produce the cord laceration so readily as does fracture of another vertebra.

Site of Fracture of the Atlas Ring.—Before passing to the mechanism of fracture, some necessary information must be obtained as to the site of the break. The reader will perhaps excuse a reference to some elementary, yet fundamental, points in anatomy. The atlas consists of two strong lateral masses which articulate above with the occipital condyles, and below with the axis. The articular facets which the lateral masses bear are very obliquely placed—the superior pair looking upwards, inwards, and backwards; the inferior pair downwards and inwards. The importance of these inclinations will be explained later. The two lateral masses are held together by two bony arches, an anterior and a posterior, so completing the characteristic ring, the long axis of which is from before backwards. The anterior arch articulates by its posterior surface in the middle third with the anterior surface of the odontoid process. The posterior arch is free, and separated from the occiput above and the spinous process of the axis below by a space, which can be only slightly lessened by extreme backward extension of the head. The bones never come into contact normally (*see* Hultkranz, Fick). The two arches are thick in the middle, where the chief ligaments and muscles are inserted, but are considerably thinner at the sides, where they join the lateral masses. This is particularly true of the posterior arch, for the groove for the vertebral artery and suboccipital nerve (sulcus arteriæ vertebralis) reduces the bone to slender dimensions at the sides. The anterior arch is considerably shorter and is stronger than the posterior, owing to the calls made upon it by the odontoid. Ordinarily both arches are fully stout enough to resist the wear and tear of shocks and movement, yet they are the weak points in the atlas ring. The lateral masses are strong and reinforced by trabeculæ, and are not likely points for fracture. That these anatomical deductions are correct may be seen from the following table, compiled from the cases enumerated in *Tables I* and *II* (gunshot wounds being omitted).

Table IV.—Sites of Fracture of the Atlas Vertebra.

Sites of Fracture	In Isolated Atlas Fracture	In Complicated Atlas Fracture	Total
Posterior arch alone	9	6	15
Anterior arch alone	2	4	6
Both arches	2	8	10
Lateral masses alone	3	2	5
Lateral masses and posterior arch	0	2	2

From this it will be seen that the posterior arch was broken in 25 cases in all, the anterior in 16, and the lateral masses in 7 only. These figures confirm our anatomical surmises.

MECHANISM OF ATLAS FRACTURE.

Fracture of the first cervical vertebra, whether in its isolated or complicated form, is so uncommon that it is evident that the forces necessary for fracture must be peculiar. In Gurlt's often-quoted series of 178 fractures of the cervical spine, there were only 6 atlas fractures, no less than 90 being of the 5th and 6th cervical vertebræ; from which it is evident that the spine is more easily broken in its lower part than in its upper. Wagner and Stolper, in their flexion experiments, were able to produce fractures of the lower cervical vertebræ only, although they specifically tried to break the atlas. It is evident that the common mechanism of fracture of the vertebræ, namely flexion and compression leading to compression of the vertebral bodies, cannot be called to account

here, for the atlas has no body. Stephen Smith unsuccessfully attempted to produce fractures of the two upper cervical vertebræ by allowing bodies to fall on their heads, whilst his remaining experiments simply demonstrate the tensile strength of the atlas and axis.

Direct violence as a cause of fracture can be ruled out as of little importance. The atlas is so deeply situated, and so well protected by muscles, that the only form of direct violence which can be relied upon to break it is a gunshot wound. I have included four cases of fracture by firearms. In number they are insignificant in comparison with the atlas wounds which the Great War must have produced. Of such, I have personally seen only one case, outlined above.

Excluding injuries by direct violence, we pass on to those produced by *indirect* means: this is by far the most interesting category, and the three possible mechanisms may be summarized as follows: (1) Fractures of one or both arches by transmitted force causing lateral spread of the bone (the odontoid perhaps broken in addition by pressure of the distorted anterior arch). (2) Fracture of the posterior arch by a crush between the occiput and neural arch of the axis, the head in full extension. (3) Fracture of the anterior arch by means of the odontoid, the head in full extension.

1. **Mechanism of Transmitted Force.**—Reference to *Tables I* and *II* shows that the common accident causing a fracture of the atlas is a fall upon the head. In one or two cases patients have been struck upon the head by a falling object, which amounts to the same thing. The essential feature of the injury lies in the transmission of force from the vertex through the occipital condyles to the vertebral column. In order fully to appreciate the paths that the lines of force must traverse, the mechanics of the injury must be discussed. The man's head on striking the ground is subjected to a force that can be approximately measured.* This force received by the cranium is finally collected at the base of the skull, and transmitted to the atlas by the occipital condyles. But since any force operates equally in opposite directions, each segment can be considered separately as suffering compression between two opposing forces. We may therefore take the skull, the atlas, the axis, and each succeeding vertebra, and consider each as being crushed by a known force. To take the first two only: in the case of the skull, some of the force is distributed to the brain and lost, and, if the cranium itself is distorted beyond its limits of elasticity, a fracture will take place, usually of the basis cranii. If no energy is dissipated through such a happening, the force passes on to the atlas in almost undiminished quantity. The occipital condyles themselves usually escape injury, presumably on account of the elasticity of the cranium upon which they are mounted. A case is, however, recorded by Sir Charles Bell, and another by Kissinger. As the atlas has no centrum, the whole of the force must pass through the planes of its articular facets, and therefore through the lateral masses, the anterior and posterior arches playing no part in its transmission. One would assume from this that fracture of the lateral masses would be the commonest fracture, but we have seen that this is far from being the case. We shall see that the reason for this lies in the inclination of the planes of the articulations on the upper and lower surfaces of the lateral masses.

In order to make this clearer, let us next consider the atlas as being compressed between two opposing forces in the same manner as we have briefly outlined for the skull. The atlas is crushed on the one side between the skull in contact with the ground and the forces operating upwards through it, and on the other side by the axis and the succeeding vertebræ representing the resistance of the body weight acting downwards. The force operating from the cranial side will necessarily be along a line at right angles to the plane of the articular surface of the occipital condyles, i.e., from above downwards and outwards and slightly forwards (*see Fig.* 373).

The resistance to the force presented by the vertebral column passes through the

*The force can be measured, if the patient has fallen from a height on to his head (as in most of these cases), by multiplying the body weight by the height fallen. Thus, an average force will be about 1400 foot-pounds, the weight being taken as 140 lb. and the height fallen as 10 feet.

atlanto-axial joints, and must therefore be charted graphically by a line running upwards, outwards, and slightly backwards (*Fig.* 373). The atlas is therefore subjected to two forces passing through it in opposite directions and on divergent lines. The crush between the occiput and axis is not a vertical one from above downwards; the lines of force diverge

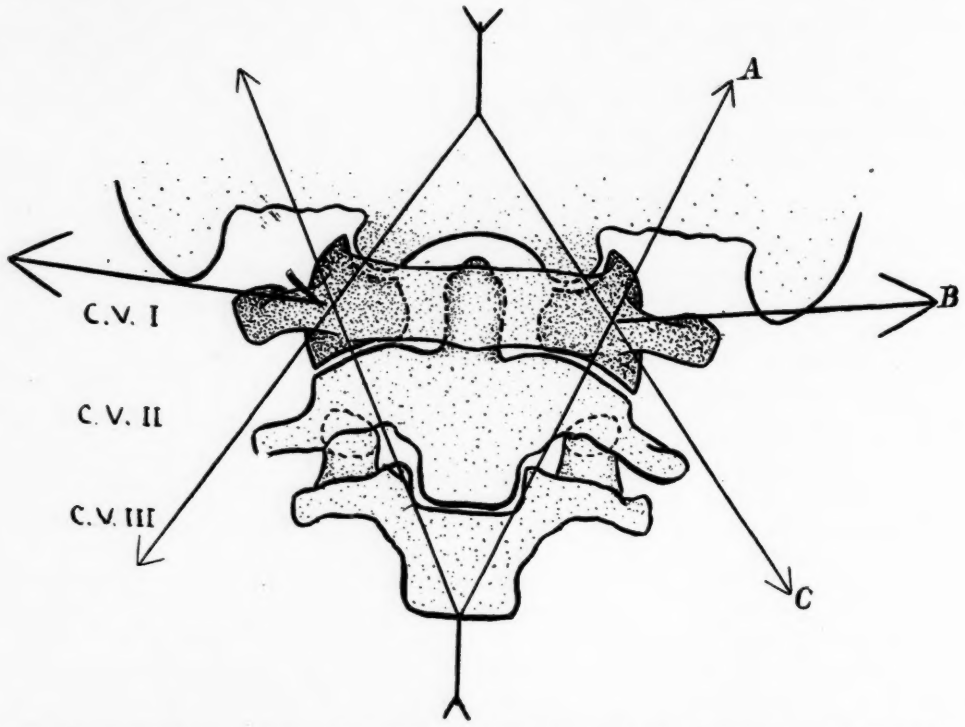

FIG. 373.—Schematic representation of the transmission of force (A and C) through the occipital condyles to the atlas, axis, and vertebral column. Note that the resultant (B) of these forces is more or less horizontal, and 'spreads' the atlas laterally.

widely, and resolve themselves, as a simple knowledge of mechanics tells us, into forces which are the mean of those already described. The direction of this force is a horizontal one, and the nett result of the crush of the atlas is, therefore, a lateral spread, a separation of the two lateral masses from one another, and a consequent tension fracture at one or more of the weak points in the atlas ring (*see Fig.* 374). This explains why the arches suffer more frequently than do the lateral masses themselves, although the latter are the paths through which the forces run.

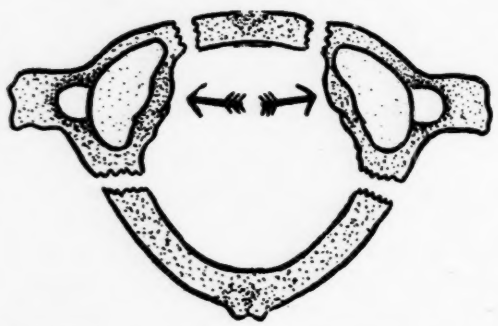

FIG. 374.—Atlas viewed from above to show the result of '⌊ spreading,' the bony arch yielding at its weak points.

The character of the injury to the atlas will vary according as the force passes equally through the two condyles, or passes in greater proportion through one lateral mass, as will occur if the head is deviated from the true sagittal plane at the moment of impact with the ground. When the head is much inclined to one side, as it must sometimes be in these accidents, a more direct compression of one lateral mass than the other will occur, with perhaps a local fracture, but even in this case a tension fracture of one or other of the arches may accompany it.

To sum up, I suggest that fractures of the atlas ring by indirect violence are tension fractures: that they are due to divergent lines of force passing through the bone, separating the lateral masses from one another, deforming the atlas into an oval with its long axis from side to side instead of from before backwards, with consequent fracture at the weak points in the ring. The posterior arch being distinctly weaker than the anterior, this will be the one which most frequently gives way on one or both sides. Individual peculiarities and predisposition to fractures may also influence the site of fracture.

2. **Fracture of the Posterior Arch by a Crush between the Occiput and Neural Arch of the Axis, the Head in full Extension.** — The articulation of the atlas with the skull depends so largely on muscles for its strength, that a sudden blow or jerk is liable to obtain a ' flying-start,' as Corner has expressed it, and the head is thrown violently backwards. When the head is in extreme extension, the occiput, the posterior arch of the atlas, and the posterior arch and spine of the axis become crowded together. Normally these can never be made to touch, but it is just possible that, with a very powerful and severe extension beyond the limits of the normal, such as would occur if a man fell from a height upon his forehead or if his head were suddenly and violently jerked backwards, the bones might come into contact, and the weak posterior atlantal arch be nipped and broken between the occiput above and the axis below. Reference to *Fig.* 370, and to Hultkranz's radiographs of the normal movements of the head (*loc. cit.*, *Plates I* and *II*) will make this clearer. Plausible though this theory sounds, it is probably not the usual mechanism ; for, although it is possible to make the bony parts in question approach one another, it is impossible to bring them into contact without dislocating the vertebræ. Nor will it account for fractures of the lateral masses.

Traction on the trunk with the head fixed spares the atlas, but may cause a low cervical fracture. Truesdell figures an excellent example of a birth injury of this nature (*loc. cit.*, *Fig.* 19). Similarly, hanging does not affect the atlas, and, as is now known, rarely injures the odontoid or transverse ligament. The usual injury is a depressed fracture of the neural arch of the axis, severing the cord (*see* Wood Jones' account of Fraser's cases, and Haughton).

A careful study of the accounts of the accidents in the hitherto reported cases affords little evidence that extreme extension of the head has been a common feature. In some cases it is definitely stated that the head was flexed at the moment of striking the ground ; in most it would appear that they were the result of falls upon the vertex cranii.

3. **Fracture of the Anterior Arch by means of the Odontoid, the Head in full Extention.**—During extension of the head, the atlas comes into close contact with the odontoid process ; and with extreme force, fractures either of atlas or odontoid might conceivably result. By the ' mechanism of transmitted force ' the anterior arch of the atlas comes into very close contact with the odontoid, owing to the deformity of the conformation of the atlas ring. It seems at any rate very possible that the atlas and odontoid may both be fractured by this means. It is very difficult to see how either of these two mechanisms (2) and (3) can come into play when the head is flexed at the time of the fall ; the secret of fracture must lie in the passage of force from skull to vertebral column. It may be noted that rotatory dislocations around the pivot of the odontoid may occur by a modification of this mechanism, taking into account the direction of the line of force and the screw nature of the articular surfaces of the atlanto-axial joints on which Fick has insisted.

SYMPTOMS AND SIGNS OF ATLAS FRACTURE.

The outstanding clinical signs of fracture of the first cervical vertebra are undoubtedly rigidity of the neck muscles and limitation of movement. The latter is naturally greater in those cases where the fracture actually involves the articular facets ; but it is a very definite feature when the arches alone are broken (as in my own case). The movement which is chiefly impaired in an uncomplicated atlas fracture is that of nodding ; but it

is to be remembered that limitation of rotatory movements of the head is not diagnostic of a lesion of the odontoid. For in fractures both of the anterior arch (owing to its articulation with the odontoid) and of the lateral mass involving the atlanto-axial articulation, rotation of the head may be restricted or impossible.

In atlas fracture, then, the patient tends to hold himself stiffly as if balancing a weight upon his head, and may support his head with his hands, particularly when making any change from the upright posture. Sir Astley Cooper's description of the patient he saw is very apt: "When he wished to examine any object beneath him he supported his chin on his hands and gradually lowered his head to enable him to direct his eye downwards; but if the object was above him, he placed both his hands upon the back of his head and very gradually raised it until his eyes caught the point he wished to see." These assisted movements, as they may be called, are suggestive of cervical caries, and only the history of a recent injury will raise the question of fracture.

In some cases a protuberance may be felt in the pharynx, at the level of the palate, painful on pressure. This is especially the case when the anterior arch is broken. In my own case of posterior arch fracture this sign was absent, nor would one have expected it. In one recorded instance crepitus has been thus detected through the mouth. More commonly the greatest tenderness is elicited by pressure either on the lateral masses in the neck, or on the posterior arch through the outer fibres of the trapezius. Reference may here be made to those curious cases in which osteomyelitis has developed and a sequestrum finally separated, being exfoliated through the mouth after rupture of a retro-pharyngeal abscess. This occurred in the case recorded by Park; but a more extraordinary case was that of Syme, where the odontoid and a large part of the body of the axis were extruded into the pharynx. Hilton records a similar case. The patients recovered.

Another symptom which not only these cases but also rotatory dislocations sometimes present is that of dysphagia and thick speech. The cases presenting it have not been closely enough examined from the neurological side to establish whether this is of central or peripheral nervous origin; it does not seem to be entirely mechanical.

Nerves.—Owing to the very close relationship between the first two cervical nerves and the posterior arch of the atlas—the one passing over it, the other under—it would be surprising if signs of injury to these nerves were not common. Of the two nerves, the great occipital, passing below the posterior arch, suffers more obviously than the suboccipital which lies upon it. This may be due to the more closely confined canal in which the former nerve lies, the latter traversing a wide groove which it shares with the vertebral artery. It must be remembered, however, that the suboccipital is mainly a motor nerve, and that any paralysis to which its injury gives rise is likely to be overshadowed by the great muscles of the neck, which are not supplied by it and are bent on keeping the broken fragments at rest. A lesion of the great occipital nerve is a great aid to diagnosis, and the presence of an anæsthesia or neuralgia in the area of its distribution should lead to a very careful investigation of the atlas vertebra. Sicard and Roger have recently drawn attention to the value of this sign, and record three cases in all of which it was present, leading them to a certain diagnosis of a lesion that would otherwise have been missed, as the *x*-ray report in one case was at first negative. In my first case there was no evidence of a nerve lesion, although it was carefully sought for. In my gunshot-wound case it was present in a degree which made the patient's life miserable. Sicard and Roger have insisted on the close relationship which the great occipital nerve bears to the posterior arch of the atlas, winding upwards round it. This they have illustrated in their paper, but their diagram is perhaps a trifle too enthusiastic, and *Fig.* 375 represents the state of affairs more accurately.

The Vertebral Artery.—This has been found compressed in one case (Delorme), and was torn by a bone fragment in a patient of Sedillot's (*see* Delorme).

Injury of the Spinal Cord.—As has already been noted, signs of cord injury, varying from a monoplegia to complete paralysis of all four limbs, were present in 19 of these 46 cases, and only 4 of these recovered. Injury to the cord is more often found in

complicated than in isolated fractures of the atlas, and this is due not only to the excessive violence which the complicated fractures imply, but also to the fact that the actual cord injury is sometimes caused by the accessory fracture, the atlas itself having inflicted no injury on the spinal cord. That injury to the various elements of the vertebral column without damage to the contained nervous elements is much commoner than has popularly been supposed is shown by J. and J. Boeckel, who collected no less than 95 cases of fracture of the spine without injury to the cord.

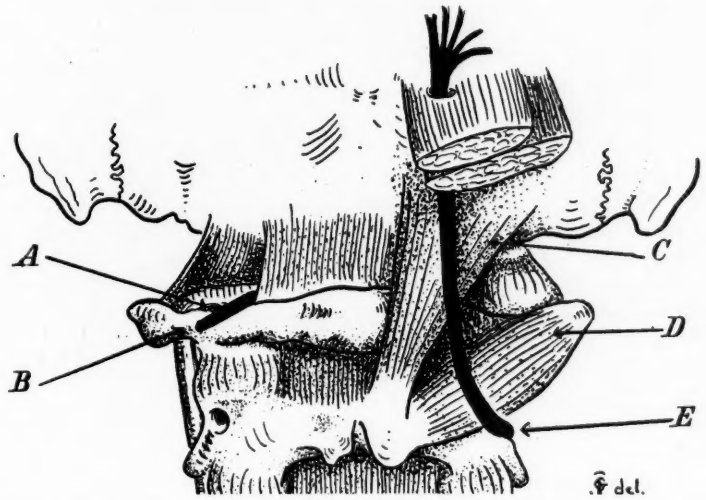

FIG. 375.—The relationship of the suboccipital and great occipital nerves to the posterior arch of the atlas. A, Vertebral artery. B, Suboccipital nerve. C, Rectus capitis posticus major. D, Inferior oblique. E, Great occipital nerve.

There are two factors at work in the case of the atlas, both tending to immunity of the cord from injury. One is the relatively large size of the neural compartment of the atlas ring ; the other is the manner of the displacement of the broken fragments. The last depends on the peculiar mechanism of fracture, which, as detailed above, tends to open out the circumference of the atlas, the fragments travelling in a centrifugal rather than a centripetal direction. It is not surprising, therefore, that the cord should escape damage as often as it does, but rather what one would expect.

DIAGNOSIS.

There is no doubt that fractures of the atlas are far commoner than the literature of the subject would lead one to suppose. The introduction of radiography has led to the discovery of injuries of the atlas and of the axis in cases where, on purely clinical grounds, the diagnosis was uncertain. There should be but little difficulty in diagnosing the condition, provided that one is always ready to suspect it, and insists on radiographs which clearly show the bone. And, greatly though radiography has helped us to establish a more certain knowledge of these injuries, the technique of photographing the upper two cervical vertebræ is difficult, and calls for very expert handling and an abundance of patience on the part of those engaged in the work. George has done much to perfect methods of *x*-ray diagnosis in this region. It is owing to the attendant difficulties that radiography, though no longer a very young science, is only now beginning to be a really reliable aid to diagnosis in injuries of the upper part of the neck. The popular belief that injuries to the atlas and axis are necessarily fatal has been another potent cause for turning suspicion away from these bones. But, given a history of a recent injury (usually a blow or fall upon the head), rigidity of the neck, limitation of head movement, and perhaps neuralgia or anæsthesia of the great occipital nerve, the surgeon should not rest until an

atlas fracture has been established or ruled out of court by perfectly sharp stereoscopic radiograms, on which the whole contour of the bone can be traced. The similarity of atlas fracture to high cervical tuberculosis has already been referred to in the text. The history of injury and the *x* ray should make the differential diagnosis simple.

TREATMENT.

Unless there are positive neurological signs of cord injury, and unless the correlation of the signs with the position of the broken fragments as seen on the *x*-ray plate leads to the belief that good can be done by laminectomy, treatment should be conservative and directed towards immobilization. In only two of the cases in this series was operation undertaken (Mixter and Osgood, and Blackwood), and both patients died. In one other case it was found at autopsy that laminectomy might have led to recovery, for here a small piece of bone had pressed on the medulla and produced gradually increasing paralysis and death. Such cases are rare. Treatment will, as a general rule, be confined to securing immobility of the head, preferably in a plaster case of the Lorenz type or in a 'Minerva' plaster, followed by a leather casket if necessary. In my own case I employed the Lorenz method, making a plaster bed by moulding crinoline wrung out of plaster-of-Paris on to the back of the patient from the top of the head to the loins. These beds are exceedingly comfortable to lie in, and make nursing much easier. Unless the articular surfaces are involved in the fracture, the functional results are good.

In conclusion, I have to thank Professor Keith for permission to publish an account of the case in the Museum of the Royal College of Surgeons, and Professor Dean for a similar kindness with regard to the specimen from the Pathological Museum of the University of Manchester.

SUMMARY.

1. Four cases of fracture of the atlas vertebra are described, and forty-two cases previously recorded in the literature are analyzed.

2. The common cause of the accident is a fall upon the head, and the probable mechanism of fracture is tension of the atlas ring due to lateral spreading of the lateral masses, owing to the divergence of the lines of force passing through the bone.

3. Two other possible mechanisms are outlined, the head being in extreme extension, but neither fits in with the nature of the accidents usually recorded.

4. Atlas fracture is by no means necessarily fatal; 45·7 per cent of the cases have recovered. When complicated by a fracture of another vertebra, the mortality is higher.

5. The commonest of such complications is a fracture of the odontoid process, next in frequency being rotatory dislocation of the atlas on the axis.

6. It is pointed out that inability to rotate the head may occur in some forms of atlas fracture. It is not pathognomonic of a broken odontoid.

7. The accident is probably by no means uncommon, but as very clear and sharp radiograms are necessary in order that the fracture may be detected, the atlas injury is often overlooked.

8. Cord injury is often absent (50 per cent). Useful aid in diagnosis may be obtained from signs of injury to the great occipital nerve.

9. Treatment will generally be conservative, directed to immobilizing the head by a 'Minerva' plaster or a Lorenz bed.

BIBLIOGRAPHICAL NOTE.

Unfortunately I have not been able to refer to the original papers of Ludloff or of Park, and have had to content myself with incomplete abstracts in other journals. In Corner's account of his isolated fracture of the atlas, the fracture is described as if it were of the axis. Corner informs me that this is a misprint. Lahey removed a bullet from within the anterior arch of the atlas, but he does not say whether there was

a fracture or not; I have not included his case. Many Continental writers refer to Stephen Smith's fifteenth case as if it were an atlas fracture. This is not so. I have traced the error to a mistake in Schmidt's *Jahrebuch* for 1872, where Smith's paper was abstracted. Most of the German writers have used this abstract without referring to the original. In neither Carson's nor Wilson's case is there convincing evidence that the atlas was broken. Kocher's *Case* 3 (*loc. cit.*) I have included, as the clinical picture is very typical of atlas fracture; but odontoid fracture cannot be excluded. Marshall's *Case* 2 (*loc. cit.*) may be an atlas fracture, but it cannot be made out in the published radiograph. It is not clear whether Marshall believes that the spur of bone is the result of fracture. This is almost certainly a normal ossification of the ligaments bridging the vertebral artery groove, and should be compared with George's *Figs.* 12, 13, 14. As for the two cases figured by George himself, the fractures cannot be made out in the published prints. Sybenga's case, whilst very interesting, I believe to be, as he himself suggests, a developmental arrest of ossification and incomplete anterior arch. The *x*-ray is an admirable one; the bony ends are clean and rounded, and do not at all resemble a fracture. There is an undoubted luxation in this case, but not a fracture.

I am greatly obliged to MM. Sicard and Roger, and M. Blanc, for sending me copies of journals I was otherwise unable to obtain.

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SHORT NOTES OF RARE OR OBSCURE CASES.

IDIOPATHIC DILATATION OF THE SIGMOID FLEXURE AND PELVIC COLON: RECURRING INTESTINAL OBSTRUCTION.

By Sir GEORGE H. MAKINS, London.

Needlewoman, age 51. For twelve months previous to her admission into the hospital the patient had suffered severely from constipation. This had been accompanied by pain, flatulence, nausea, and occasional vomiting, while distention of the abdomen had been steadily increasing.

Sept. 1, 1904.—On admission, the patient was not suffering greatly, the temperature and pulse were normal, the tongue moist and only lightly furred, and the general condition fair. No sufficient action of the bowels had occurred for some days. The abdomen was much distended, especially in the lower segment, and slow peristalsis was visible.

The patient remained under observation for a month, during which period numerous mucous casts were observed in the stools, and the latter were deficient both in bulk and consistence in spite of the regular administration of purgative medicines and enemata. The degree of distention of the intestines varied; at times the abdomen became flaccid, but peristalsis was generally visible. Vomiting occurred at intervals.

On Oct. 1 the abdomen was opened through the left rectus muscle, and the dilated sigmoid loop at once presented. No twist or other source of mechanical obstruction was discovered. A large quantity of gas and a small amount of fæcal matter were evacuated by means of a trocar and cannula. A lateral anastomosis was established at the base of the loop, the latter was returned into the abdomen, and the cavity closed.

The patient made an uneventful recovery, and left the hospital in three weeks' time, the bowels acting fairly well.

In December, 1905, thirteen months later, the patient returned to the hospital in much the same condition in which she had first presented herself. Constipation and flatulence had been gradually increasing for some months, and on admission the abdomen was distended as before, peristalsis in the dilated sigmoid loop being visible and constant.

The abdomen was reopened through the old scar, and the loop was delivered. The latter was twisted on itself at its base, and the anastomosis opening completely occluded. The gut was punctured and its contents evacuated, clamps were applied, the loop was removed, and the free ends of the sigmoid and pelvic colon were closed about two inches from the anastomotic opening. An uneventful recovery again followed, but at times some abdominal distention accompanied by peristalsis was noticed.

In September, 1906, eight months later, the patient returned in exactly the same condition. In my absence from London, Mr. Nitch kindly opened the abdomen for me. A large volvulus consisting of the unaltered descending colon, and a greatly distended pelvic colon, was discovered extending upwards into contact with the right lobe of the liver. Mr. Nitch untwisted the loop after evacuating its contents with a trocar and cannula, and anchored the loop to the anterior abdominal wall with a view to preventing the occurrence of a subsequent twist. The patient again improved, but difficulty was experienced in keeping the bowels acting, and six weeks later I opened the abdomen again, increased the length of attachment of the two limbs of the anastomosis, and added some further ventral fixation stitches to the abdominal wall. The patient left the hospital fairly well, only to return three months later, again suffering from almost absolute obstruction.

The abdomen was opened for the fifth time, the bowel relieved by puncture, and as there seemed some doubt whether the fixation stitches had not taken part in the production of the obstruction, the bowel was once more freed from its attachment to the anterior abdominal wall.

During the next twenty months the patient got on fairly well, but on Dec. 19, 1908, she returned with complete obstruction. On this occasion the loop was punctured, and, when emptied, the wall was freely plicated so as to diminish the capacity of the sac formed by the pelvic colon. In July, 1909, and again in November, the patient was admitted suffering with subacute obstruction. On these two occasions she was treated with purgatives and enemata, and sent out relieved.

FIG. 376.

On the evening of April 25, 1910, she came for the last time; she had had no action of the bowels for three days, and was vomiting. In view of past experience it was not considered necessary to operate at once, and an enema was ordered to be administered; during this procedure an attack of syncope occurred and the patient died.

The subsequent post-mortem examination confirmed in every respect what had been noticed at the numerous operations. Little or no alteration had taken place in any part of the colon above the junction of the descending portion with the sigmoid flexure, the bowel wall being neither thickened nor dilated. The upper limb of the anastomosis retained its form as definitely as upon the day on which it was established, the dilatation being limited to the distal limb. The accompanying illustration, *Fig.* 376, is a drawing made from a photograph taken at the time of the post-mortem examination, but it hardly gives a correct impression of the immense balloon formed by the pelvic colon and upper part of the rectum. No source of obstruction was discovered in the lower rectum or anal canal. A remarkable fact disclosed by the post-mortem examination, also confirming previous observations, was the entire absence of any peritoneal adhesions either between adjacent coils of intestine or between intestine and abdominal wall. Even the sites of the stitches inserted for the colopexy and the coloplication were free and practically invisible. Considering the numerous operations, accompanied on each occasion by puncture of the bowel to evacuate its contents, this absence of any evidence of infection and subsequent inflammation was very striking.

SUMMARY.

The main points of interest in this case lie, first, in the development of the secondary dilatation and its limitation to the pelvic segment of the colon; and secondly, in the fact

that the whole repertory of methods of treatment (short-circuit, excision of the loop, colopexy, and coloplication), with the exception of the provision of a permanent vent-hole, proved unsatisfactory. It was intended on the last occasion to establish a permanent fistula as was done by Dr. W. B. Coley, but this procedure was prevented by the sudden death of the patient.

The limitation of the original dilatation to the sigmoid colon is not uncommon, and is plausibly explained in many cases by the formation of a kink at the junction of the lower limit of the flexure with the pelvic colon. The remarkable feature lies in the development of the secondary dilatation equalling in capacity the original loop, and it may be of interest to consider how this may be explained. It appears to be clear that the case belonged to the category of colic neuroses from the first, the main characteristic of the obstruction on every occasion being the accumulation of large quantities of flatus and small quantities of liquid fæces. Such distention is common in the subjects of general neuroses, while its acute varieties are seen in the acute dilatation of the stomach and the intestinal distention associated with fractures of the spine accompanied by injury to the spinal cord. I am inclined to refer the local dilatation to a local defect in the nerve system governing the intestinal wall. Another explanation has been suggested in the nature of the primary anastomosis established, the communication being of the antiperistaltic type. In regard to this theory, it may be pointed out that the segment which usually suffers in an unsatisfactory anastomosis is the proximal; thus, after an axial union of divided small intestine, it is the proximal segment which becomes thinned and dilated.

A CASE OF OSTEITIS DEFORMANS (OSTEOPOROSIS, OSTEOMYELITIS FIBROSA), WITH PRONOUNCED AFFECTION OF FOREARM.

By HERBERT FRENCH, London.

The following case of osteitis deformans is of special interest from the fact that one of the most marked of the deformities is in the left forearm—a part which is not commonly affected to this extent.

The deformity is shown in the photograph (*Fig.* 377), which, however, indicates the appearance less strikingly than was the actual case. The changes in the left radius are shown in the skiagram (*Fig.* 378), and the changes in the right femur are shown in the skiagram in *Fig.* 380. Skiagrams of normal bones are given in *Fig.* 379 and *Fig.* 381 for comparison.

There were similar, though very much slighter, changes in parts of both tibiæ. No skiagraphic abnormalities were found in connection with the foot bones, pelvic bones, spine, ribs, scapulæ, clavicles, sternum, humeri, or skull; although, from the patient's own account of his symptoms, it seems likely that early changes are beginning in the ilium, spine, and skull.

The first bone involved appears to have been the right tibia, and the second the left radius; and the purpose of this note is to

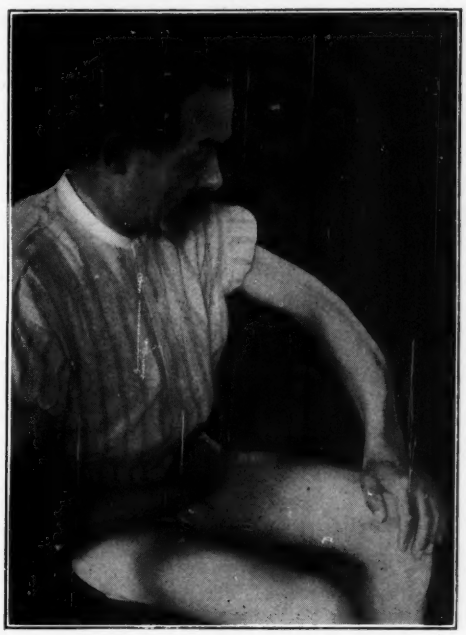

Fig. 377.—Photograph of the patient, showing the bending and deformity of the left forearm similar to that which might result from a badly united fracture at the junction of the middle and distal thirds of the radius.

record the pronounced radial changes, which are unusual. The appearance of the patient's forearm at the present time is such as might result from the effects of a badly-united fracture of the left forearm at the junction of its lower and middle third.

French authorities have recorded a positive Wassermann reaction in many cases of this disease, but in the present case the Wassermann test, carried out by Dr. Eyre in the Bacteriological Laboratory at Guy's Hospital, was negative, and the primary cause of the bony lesion was not ascertained.

The patient was sent to me in the first instance by Dr. Groves, of St. Ives, Huntingdonshire, to whom I am indebted for permission to publish these notes.

The patient himself has described his subjective sensations, as the disease progressed, during the last ten years. The following extracts are taken from this description :—

FIG. 378.—Skiagram of the patient's left radius and ulna, showing typical osteoporosis of the radius.

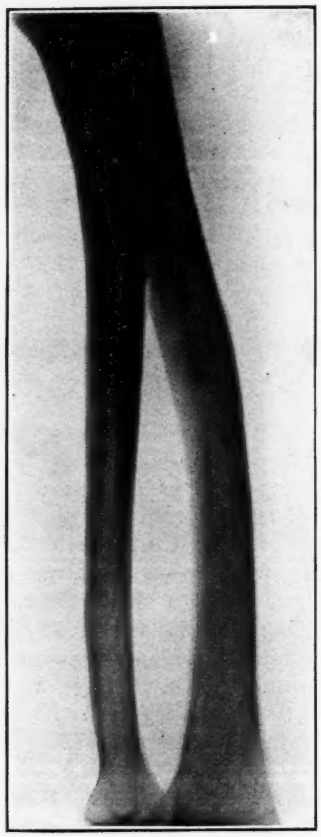

FIG. 379.—Skiagram of the patient's normal right radius and ulna for comparison with *Fig.* 378.

FAMILY HISTORY.—Grandparents healthy and lived to about 80. Mother died of apoplexy at 65. Father at 78 ; he was very poor in early life, and worked hard until his 76th year. He died of atheroma of heart and arteries. Brothers and sisters all alive. No history of still-born children. No consumption. No cancer or bone troubles amongst them.

PERSONAL HISTORY.—Age 50. Never had typhoid, nor lived in a foreign country. A life-long abstainer and non-smoker. About 1907 felt occasional stabbing pains in right tibia, with a sensation in the leg as if it would give way. The pain induced an unconscious limp.

Pains gradually became more frequent, and were supplemented by an almost continuous dull ache. The ache increased as if the bone were being gripped tightly. There was swelling of the ankle at night. The doctor discovered alteration of bone, and prescribed mercury ointment, bandaging, rest, Bier's method, and suitable medicines. This having no effect, a periosteal incision was made.

While in bed, slight curvature of the left radius was noticed late in the year 1909. About a year later the disease appeared in the right femur, as shown by *x*-ray examination. The bone was found to be thickened, and probably becoming less dense. The condition has progressed steadily. The radius has curved more. The tibia has enlarged and curved—although removal of bone by the trephine checked this curvature. The femur has curved.

All the curvatures correspond in form. The knee and ankle-joint give considerable trouble, and hurt at times. Occasionally there is an ache and tenderness at the head of the femur, and the top of the ilium is quite tender. The tibia is always more or less hot and tender, and there is pretty constant aching in both tibia and femur. Pain is frequent, intermittent, and varies in character.

FIG. 380.—Skiagram of the lower part of the patient's right femur, showing typical thickening aud undue transparency from osteitis deformans (osteoporosis).

FIG. 381.—Skiagram of a normal femur for comparison with *Fig.* 380.

There are stabbing pains, waves of ache and pain along the bones, smarting and burning, pricking and boring sensations, with a 'trying-to-expand-in-a-resisting-substance' feeling. Pain is not confined to any particular time of day, nor affected by the weather. Back gets tired, and there is aching across the loins. All the symptoms are aggravated by activity.

The radius suffers in the same way. Pronation and supination are increasingly affected. The disease appears to be now starting in the left tibia. There is a feeling as if the bone were expanding, but no alteration can be detected. Sometimes on walking there is pain in this bone, and there is some actual tenderness occasionally.

Frequently, of late, patient has had a 'bad' head, which is relieved by rest and sleep —if he can sleep.

REVIEWS AND NOTICES OF BOOKS.

On Gunshot Injuries to the Blood-vessels. By Sir George Henry Makins, G.C.M.G., C.B., President of the Royal College of Surgeons of England. Super roy. 8vo. Pp. xii + 251, with 4 coloured plates and 60 figures. 1919. Bristol: John Wright & Sons Ltd. 21s. net.

We have nothing but praise for this monumental work of Sir George Makins, and it should find a place in the library of every surgeon. The earlier pages consist of an amplified and revised edition of several papers on gunshot injuries of the blood-vessels which appeared under his name in various periodicals during the late war, and the whole book is a veritable storehouse of information upon everything pertaining to vascular injuries, their effects, and their appropriate treatment.

The perusal of the pages of this contribution from the pen of so distinguished an authority upon injuries to the blood-vessels cannot fail to impress the reader with the painstaking care and scientific spirit with which the data contained therein have been weighed, sifted, and worked into the text.

As we are told in the introductory chapter, the chief information gained during the late war regarding the treatment of vascular injuries consists in: (1) A more accurate knowledge of the signs of arterial injuries, such as may be gleaned from an investigation of the peripheral and vaso-motor nervous system, and from auscultation both over the injured artery and over the precordial area; (2) The actual mode of development of some of the consequences of arterial damage; (3) The effects, by obliteration of the main vessels, on the vitality of the part supplied by the peripheral branches affected; (4) An increased knowledge of the exact nature of the anatomical lesions of injured vessels; (5) The arrangements of the vessels in relation to the various forms of traumatic aneurysm; and (6) Increased knowledge as to the relative advantages obtained by obliteration of the main vessels by ligation, by simultaneous occlusion of artery and vein, and by efforts to maintain their viability by suture.

Of particular interest are the arguments, based on clinical and experimental observation, brought forward in support of simultaneous ligature of both vein and artery in injuries in which the necessity of tying the artery arises; this practice became almost the universal rule in the latter part of the campaign.

The author's remarks upon the suture of wounded vessels are worthy of consideration; he points out that whereas it had been hoped at the beginning of the war that abundant opportunity would be afforded for practising the various methods of arteriorrhaphy, the severe sepsis, and the unsuccessful efforts to control this during the early part of the campaign, effectually prevented any extensive use by British surgeons of this method of dealing with wounded blood-vessels. In the latter eighteen months of the war, with more thorough excisions and the more successful control of sepsis, the number of cases of arteriorrhaphy increased.

Our experience coincides with that of Sir George Makins, that it is unnecessary in the technique of vascular suture to employ the very small needles and the very fine silk which had been advocated by Carrel; Japanese silk, and even thread, of less fine size, and needles of larger calibre, are perfectly compatible with successful results. He mentions the common carotid, the external iliac, and the common femoral arteries as those in which arterial suture is most strongly called for.

The author emphasizes the evil reputation which attaches to injuries of the popliteal vessels, and he notes that gangrene occurred in 45 (8 per cent) of the cases collected and investigated by him.

The evidence which he has so carefully sifted appears to favour arteriorrhaphy as the method of choice, especially if the wound be lateral; the relative good results obtained from the use of a Tuffier's tube are also emphasized, and he insists that if ligature is employed, the vein must be tied as well as the artery.

The bad results of injuries to the tibial vessels are only too well known, to those, at least, who have worked in France; and it is pointed out that the number of deaths which have occurred, consequent on wounds of these vessels, is quite incommensurate with the size of the arteries involved.

Very valuable information is forthcoming in connection with injuries to the carotid vessels and their cerebral complications. It is emphasized that in the case of injuries to these vessels the risk of delay in active interference is far less than that attendant on the sudden occlusion of the carotid artery at a period when the general blood-pressure is low.

The book will be the standard work of reference for many years to come for writers on the surgery of the blood-vessels; it is one of the most remarkable productions in the medical literature of the war. The beautiful illustrations are a feature of the work.

Menders of the Maimed. The Anatomical and Physiological Principles underlying the Treatment of Injuries to Muscles, Nerves, Bones, and Joints. By ARTHUR KEITH, M.D., F.R.S., Conservator of the Museum, and Hunterian Professor, Royal College of Surgeons of England. Demy 8vo. Pp. xxi + 335, with many portraits and 7 figures. 1919. London: Henry Frowde and Hodder & Stoughton. 16s. net.

SELDOM does it fall to the lot of the reviewer of surgical literature to have so pleasant a task as the perusal and description of such a work as this.

The author, a medical graduate of our most northern University of Aberdeen, and now Hunter's sucessor in London, takes his reader many a pleasant journey to the various seats of learning in this country and on the Continent, and tells in simple language the story of discovery of principles and initiation of practice in the science and art of healing as applied to deformed and injured limbs.

As the alliteration of its title would lead us to expect, the work is written in so popular a style that it may be taken up in an odd moment of leisure by the most tired student and used as pastime and refreshment; but nevertheless the keenness of scientific accuracy does not suffer by this popular method of treatment.

Hunter, Hilton, and Thomas are the first three subjects of discourse. Hunter recognized that, at a certain stage in its repair, a broken bone would mend more quickly if it were put into use, and he taught that the patient's will should be made to help in muscle re-education. Hilton's doctrine of rest and pain is described, together with his view that surgery was merely an adjunct to the healing power of nature. The picture of H. O. Thomas is particularly happy as showing how the busy general practitioner, coming of a race of bone-setters and without any knowledge of pathology, has nevertheless invented apparatus and applied principles which stand unrivalled to-day in the treatment of disease of the joints.

Then follow a number of chapters in which the various principles of treatment of deformities are illustrated by the lives and teachings of their pioneers. Little seeking cure of his own club-foot and submitting to tenotomy by Stromeyer; Adams accurately dissecting and describing club-feet; Marshall Hall demonstrating the relation of the spinal cord to nerve and muscle reflexes; Duchenne armed with his faradic battery; Beevor and Sherrington discovering the relations between the central nervous system and the muscles; Waller, His, and Head and their researches on nerve function, injury, and repair; tendon transplantation from its simple forms up to its most modern development in the cinematization of stumps; Lane with the introduction of internal splints for fractures; Jones advocating external splints with traction for the same injuries; American orthopædists in relation to deformities of the spine, including Abbott's treatment of scoliosis; the gospel of movement preached by Lucas-Championnière, and, alas, so soon forgotten; the battle of the periosteum and the work of Ollier and Macewen—all these varied subjects are dealt with in the form of a fascinating story and yet with true critical discrimination.

Perhaps the most notable omission in the series of articles is that of the treatment of fracture by weight extension of which the late Professor Bardenheuer was the pioneer.

The work is so vivid in its interest that it is hard to believe it to be the production of a mere anatomist, and if Professor Keith is not already practising as an orthopædic surgeon we feel sure that when he does so he will have as great success in this rôle as he now has in that of a teacher and author.

Nerve Injuries and their Treatment. By Sir J. PURVES STEWART, K.C.M.G., C.B., and A. EVANS, M.S. Demy 8vo. Pp. xii + 249, with 137 illustrations. Second edition. 1919. London: Henry Frowde and Hodder & Stoughton. 12s. 6d. net.

THE treatment of injured nerves formed an important feature in the surgery of the recent war, and much still remains to be done for the sufferers. The influence of the intensified attention paid to these injuries will have a considerable effect upon civilian practice in the future. Many cases of nerve injury remained untreated in the past, owing to a lack of knowledge regarding the special methods of diagnosis and treatment required. In this connection the second edition of Stewart and Evans' *Nerve Injuries*, which has been largely rewritten in the light of further experience, will be useful and interesting. The general practitioner will find in it much to assist him in the diagnosis of a difficult class of cases, and surgeons will obtain many practical hints.

He who would operate on cases of nerve injury requires an intimate knowledge of anatomy, an inexhaustible fund of patience and readiness of resource, in addition to the skill of an adept surgeon. The anatomical descriptions in this volume are short, but definite. They direct attention to special points not always found in anatomical text-books, and teachers of anatomy will receive encouragement in their endeavour to show the importance of abnormalities in the distribution of nerves, which students are so prone to ignore. The diagnosis and prognosis of an important case may depend upon an abnormal nerve, and nothing can be done until the possibility of its presence is remembered, or discovered by consultation. The authors point out that the occasional anastomotic branch between the ulnar and median in the forearm may carry all the motor fibres of the ulnar. Hence a lesion of the ulnar, above the level of junction, causes little or no motor impairment in some cases. They might also have added that, on the other hand,

fibres passing from the ulnar to the median enable the flexor sublimis, in some instances, to flex, at the proximal interphalangeal joint, the ring and little fingers, and to a slight extent the middle finger, after division of the median nerve in the arm. Other anatomical abnormalities play their rôle in nerve compression. The symptoms caused by a cervical rib, and by an elongated transverse process of the seventh cervical vertebra, are mentioned. Reference might be made in a subsequent edition to the pressure effects sometimes caused by the first (dorsal) rib, as described by Stopford and Telford in the October number of this journal. A valuable warning is given not to weary the patient by too prolonged examination, and simple methods are advised as tending to more accurate responses; but no mention is made of the advantage of attending to the temperature of the affected limb during examination. Burrow and Carter use a paraffin bath at 40° C., as they found that the electrical and sensory tests were unreliable when applied to a cold limb.

Diagrams of motor points and of the areas of nerve distribution, together with numerous illustrations of the effects of nerve injury, greatly help to elucidate the text. A description of operations on nerves in general is followed by particulars relating to special nerves. The necessity of treatment, before and after operation, to prevent the development of various deformities, is rightly insisted upon, and directions are given for carrying it out.

Amongst so much that is good a few omissions may be noticed. Partial resection is described for cases of incomplete division of nerves, but its disadvantages are not indicated. Much controversy centres around the advisability of anastomosis or implantation of nerves, but this fact is not mentioned; although the authors give particulars of an operation for gunshot injury of the fifth cervical root in which it was not possible to find the proximal portion, and the spinal accessory nerve was used for anastomosis. "Six months later the patient could voluntarily contract his biceps muscle."

More space might well have been given to the treatment of cases of irreparable nerve injury by means of tendon transplantation and tendon fixation. This method promises to attain considerable importance in the near future. A recent addition has been made by Fenwick's transplantation of slips of the temporal muscle to reinforce the orbicularis palpebrarum and the orbicularis oris in certain cases of facial paralysis.

A Treatise on Orthopædic Surgery. By ROYAL WHITMAN, M.D., F.A.C.S., New York. Pp. xii + 914, with 767 illustrations. Sixth edition. 1919. London: Henry Kimpton. 36s. net.

THE author, from his leading position as the best-known orthopædic surgeon in New York, represents the views of American orthopædic surgeons in an authoritative manner.

Orthopædic surgery has been recognized as a separate entity in America much longer and more thoroughly than in this country. And even in America the recent epidemics of poliomyelitis and the crippling accidents of the war have made this branch of surgery more important than ever.

The book deals with injuries and diseases of the bones, joints, muscles, and nerves in a systematic manner, and the standard methods of treatment are clearly and critically described. The author presents the various newer methods, i.e., Albee's spinal graft and Abbott's treatment of scoliosis, with fairness; and he shows his own appreciation of their drawbacks and limitations. The chapter on military orthopædics is too brief to be of much value if taken by itself, but as an appendix to the rest of the volume it is decidedly useful.

The description of various splints and orthopædic appliances, together with the clear account of their method of application and the critical discussion of their relative values, constitutes the chief value of the book to the English reader.

United States Army *X*-Ray Manual. Demy 8vo. Pp. 506, with 219 illustrations. 1919. London: H. K. Lewis & Co. Ltd. 18s. net.

THIS book is compiled under the direction of the Surgeon General of, and is the official handbook for radiologists in, the United States Army. As it was only published in 1919, it is rather belated from a war point of view. It is confined almost entirely to the use of *x* rays in the army, and as such is a useful handbook. For a work of its size it would seem to us that too much space has been devoted to the physical side of *x* rays. A radiologist under Service conditions is not particularly interested in this aspect of the question, while for a general work the information given is meagre. The chapters on apparatus are good, and some very useful diagrams on the wiring of an *x*-ray installation are included. A great deal of attention is devoted to the localization of foreign bodies. Most of the methods in vogue in the British Army are explained, while some of the more complicated methods involving the use of Hirtz's compass are reviewed. The chapter on localization of foreign bodies in the orbit is excellent. There are some useful chapters on the examination of bones, and a description of the more common pathological conditions met with. The diagrams of the outline of cavities of the heart are of value in examining that organ. Some good reproductions of barium meals are given, but it is impossible to discuss such a subject in twelve pages of letterpress. *X*-ray therapy is dismissed in sixteen pages. It is an excellent manual, but seems to us to have attempted far too much in such a limited space.

Données Actuelles sur la Régénération Osseuse Aseptique chez l'Adulte. By Dr. JEAN HAOUR, Interne des Hôpitaux de Lyon. Pp. 126, with 55 figures. Lyon: Imprimeries Réunis.

THIS monograph represents one of the most recent attempts to defend the Lyons teaching about periosteal regeneration. On the whole it is well and fairly written, and deserves close attention by all who are interested in bone surgery.

It is evident at the outset that the school which the author represents is fighting a losing battle, or is executing a strategic retreat in order to save something of the principles which they once taught. Clearly the majority of French surgeons have found by practical experience that total or even large esquillectomies produce very bad results, and that the periosteum of which they expected so much has given them very little. Blake, Heitz-Boyer, Alquier, and Tanton are quoted as having had this experience, and against them are placed Leriche, Durand, Cotte, Desgouttes, and Delore, who have had good results from esquillectomy. In 1917, and again in 1918, a warning against the practice of primary esquillectomy was issued to all French military surgeons.

Dr. Haour commences with the proposition that total subperiosteal esquillectomy in aseptic conditions will result in osseous regeneration. But by the time he has reached his conclusions, he qualifies this by plainly stating that the periosteum is a fibrous tissue without osteogenetic properties, and he is content with claiming that it is possible to remove a layer of bone particles with the periosteum from which regeneration will ensue. And again, in the very middle of his thesis he seems frightened by his own proposition, for he complains that surgeons, not content with performing large esquillectomies, have carried out total esquillectomies and so produced conditions of non-union. But it is just this total esquillectomy which is the matter of controversy, and surely if the periosteum with its adherent bone granules can reproduce the bone, then there need be no reason to fear the result of the removal of all the loose fragments.

The degree of regeneration which follows the removal of bone fragments depends upon the preservation of bone granules connected with the periosteum. The author gives several diagrams illustrating this point, but it is unfortunate that this cardinal factor could not have been illustrated by actual microphotographs from experimental cases. The raised periosteum is represented as having a layer of small separate bone granules adherent to its deep surface—a condition of things of which, except in an inflamed bone, one may be allowed to doubt the possibility.

Perhaps the most arresting passage in the book is that in which with a few sentences he summarizes the results which have occurred in the French and English armies. According to official figures he states that in 1919 there were 2000 cases of pseudarthrosis in the French army, and he goes on to say, "whilst in the English army cases of absence of consolidation will be still more frequent." In English military surgery, it is said, profuse suppuration is habitual and this results in subsequent elimination of necrosed fragments and non-union of fractures. Such statements need only be quoted in order to show to what lengths the apologists for primary esquillectomy have been driven. It is not possible to obtain official figures about the total number of ununited fractures in the British army at any one time, but enough can be ascertained to show that Dr. Haour's estimate is incorrect. In the early part of 1919 there were not quite twenty large centres to which all ununited fractures were sent. Enquiry at the largest of these elicited the information that there were less than twenty cases at each centre, but taking each figure at the maximum, this would only give 400 cases, as compared with 2000 in the French army! And further, the great majority of these cases, probably 80 per cent, were cases resulting from loss of bone, either by the original injury or by primary esquillectomy. The actual number of cases of pseudarthrosis due to non-intervention and to mere 'profuse suppuration' with late elimination of necrosed fragments has been very small indeed.

The experimental contributions to the subject are hardly worth serious consideration. The experiments are very few in number, and the final results are not shown, because unfortunately in the retreat of 1918 they were lost. The best result shown (*Fig.* 9) is that of the radius of a rabbit, where, after subperiosteal resection of a few centimetres, continuity of the shaft is re-established thirty-three days later. But in this case, definitely stated to be that of an adult animal, the epiphyses are clearly seen, and therefore growth from these centres cannot be eliminated. In other cases regeneration falls very far short of restoration of continuity.

The clinical cases brought forward are also instructive as showing how very far short of normal regeneration are the results which the most approved methods of subperiosteal resection can give. *Fig.* 16 represents an elbow after primary excision, but certainly there is nothing approaching a reconstitution of the resected parts. In *Fig.* 48 there is represented a femur in the shaft of which there has been a total esquillectomy. But it is clear that one large bridging fragment has been left, round which regeneration has occurred. In *Figs.* 49 and 50 there is shown a femur after a genuine total esquillectomy; but although in the latter figure consolidation has taken place, evidently this has been accomplished by the main fragments having been drawn together. and not by the filling of the gap with new bone.

The monograph is of great interest and value as showing the weakness of the position of those who still pin their faith to periosteal osteogenesis, and who advocate primary esquillectomy for comminuted fractures.

The Art of Anæsthesia. By PALUEL J. FLAGG, M.D., Lecturer in Anæsthesia, College of Physicians and Surgeons, New York. Second edition, revised. Large 8vo. Pp. xviii + 367, with 136 illustrations. 1919. London: Lippincott Co. 18s. net.

Handbook of Anæsthetics. By J. STUART ROSS, M.B., F.R.C.S.; with an introduction by H. ALEXIS THOMSON, C.M.G., M.D.; chapters upon Local and Spinal Anæsthesia by W. QUARRY WOOD, M.D.; and upon Intratracheal Anæsthesia by H. TORRANCE THOMSON, M.D. 8vo. Pp. 205, illustrated. 1919. Edinburgh: E. & S. Livingstone. 7s. 6d. net.

THE field of anæsthesia has been so fully covered by text-books like Hewitt's in this country and Gwathmey's in America, and so little has been added of late years to our knowledge, that perhaps the best that can be hoped for in a work on the "art of anæsthesia" is that it shall present to the student the subject matter in a clear, simple, and sufficiently exhaustive way, and the book may be said to justify its existence.

The fact that Dr. Flagg's *Art of Anæsthesia* now appears in a second edition presumably shows that it meets a demand among medical students and practitioners, especially in America. The author enumerates and discusses in detail every practical point connected with the induction and maintenance of, as well as the recovery from, the anæsthesia produced by each of the principal anæsthetics in turn, namely, nitrous oxide, ether, ethyl chloride, in sequence or in combination.

Ether, either alone or with nitrous oxide, is of course the anæsthetic of choice all over America, and most attention is bestowed on this drug. As regards chloroform, the author is in full agreement with the finding of the Committee of the American Medical Association of 1912, that "the use of chloroform for major operations is unjustifiable," and that "for minor operations its use should cease." He would even abolish its employment in obstetrics. While we believe this is good counsel and agrees with our own practice, we think that the dark picture of the results of chloroform-giving is out of proportion and perspective to the actual facts. The chapter on carbon dioxide and re-breathing is interesting, and should be read by the anæsthetist who has puzzled over apnœa in a patient while under the open-ether mask; it is shown to be due to acapnia, that is, an absence of carbonic acid gas.

The book is well printed and the illustrations are good, but we confess that the author's fondness for classification, division, subdivision, and tabulation make the reading rather trying and often not a little tedious.

A much smaller and less pretentious work is the *Handbook of Anæsthetics* by J. Stuart Ross, but it is one more likely to appeal to the student. It is clear and concise, and for all practical purposes exhaustive; and more than this, it is interesting reading—quite uncommonly so. The first two chapters, on the physiological action of anæsthetics and shock, are lucid and condensed explanations of the physiology of anæsthesia and Crile's theory of shock, and his methods of prevention. We think they might be read with profit by many operating surgeons. That in general anæsthesia "part of the brain only is asleep," and that although the patient does not consciously feel pain, yet "injury to nerves still causes impulses to be transmitted to the brain," bringing about changes in the cells of the grey matter and the more evident alterations in blood-pressure and respiration, are reminders much needed by both surgeon and anæsthetist.

The author deals at some length with the newer methods of administering nitrous oxide gas and oxygen for prolonged anæsthesia. He evidently favours ether, especially by the open method, as the anæsthetic for choice in prolonged administrations; but he is not unduly prejudiced against chloroform, or mixtures of chloroform and ether, if the anæsthetist but fully realizes the fact that in chloroform he is dealing with a dangerous drug, and brings to its use a greater care and a livelier sense of added responsibility.

The author makes very sound observations on the limitations of the more unusual methods of giving anæsthetics, namely, intratracheal and rectal. The accidents and sequels attending the giving of anæsthetics are dealt with shortly but sufficiently. The chapters on spinal and local anæsthesia by colleagues are of more interest to the surgeon than to the anæsthetist, but deal both clearly and fully with their subject matter. The volume is well illustrated and the type clear. It is an admirable little book, and much to be recommended to the profession.

The Urethroscope in the Diagnosis and Treatment of Urethritis. By MAJOR N. P. L. LUMB, O.B.E., R.A.M.C. (T.C.): A Contribution to Urethroscopy. 8vo. Pp. xii + 51, illustrated by 40 original illustrations. 1919. London: John Bale, Sons & Danielsson. 10s. 6d. net.

As a record of conditions detected by the author by means of the urethroscope this little book is admirable. As a guide to the use of the urethroscope in urethritis it is disappointing. Wyndham Powell's instrument is adequately described, but Buerger's instrument is only mentioned, whereas it is obvious that many of the pictures were obtained with the latter instrument. The directions for introducing the instrument are insufficient for a beginner. The author writes of introducing the tube like an ordinary metal sound. It is preferable to introduce the tube and obturator just within the meatus, and to carry out the further passage of the tube under vision and air inflation. By this means the preliminary passage of the tube does not spoil the picture. The coloured plates are beautifully produced; the normal urethra is first shown, and then various pathological condi-

tions, notably peri-urethral abscess, inflammation of Littré's glands (is it too late to protest against *Littritis* ?), and infiltrations. The effect of treatment of these conditions is well shown.

Under "Indications for Urethroscopic Examination in Urethritis," the use of the instrument is advocated in cases of acute urethritis to determine when treatment may be stopped. Surely this is unnecessary, and impracticable as a routine. Under the same heading various pathological conditions are mentioned, but for their treatment we are referred to works on the general treatment of urethritis.

The author, in the preface, states that he hopes to expand the work, when doubtless further details will be given.

Injuries to the Head and Neck. By CAPT. H. LAWSON WHALE, M.D., F.R.C.S., R.A.M.C.; with a Preface by Col. F. F. BURGHARD. Demy 8vo. Pp. xii + 320, with 130 illustrations. 1919. London: Baillière, Tindall & Cox. 15s. net.

IT is unfortunate that a book which contains no reference to fractures of the vault and base of the skull with their many intracranial complications, concussion, subluxations of the spine, and birth injuries of the brachial plexus, should masquerade under the title, "Injuries to the Head and Neck." Colonel Burghard, in his preface, refers to "the surgery of gunshot injuries of the face and jaws such as this volume deals with," and relegates it to its proper position amongst surgical literature. It is essentially the product of a nose, ear, and throat specialist dealing with the terrible injuries inflicted on those special regions in modern warfare, with occasional incursions into realms of more general surgical interest. Unfortunate references to 'gastrostomy' are made, when the sphere of general surgery is encroached upon, as if the operation of 'jejunostomy' had never been invented. In his own domain the record of the writer's views may be of use to those who have to assume similar responsibilities in future wars, whilst his personal collaboration in injuries of the regions to which he has devoted special study would certainly be enhanced in value by the experience here recorded and by the enthusiasm which he has brought to bear on their study.

SHORT NOTES ON BOOKS.

Surgical Nursing and After-treatment. By RUTHERFORD DARLING, M.D., M.S., F.R.C.S. Pp. xii + 582, with 129 figures. London: J. & A. Churchill. 8s. 6d. net.

THIS is a very compact and well-written book intended for nurses and dressers. It includes a simple account of all ordinary surgical diseases and operations, and is illustrated by clear diagrams. A good description of theatre technique is included, and there is a useful appendix dealing with lotions, drugs, and the common terms used in prescriptions.

Essentials of Surgery: a Text-book for Student and Graduate Nurses. By ARCHIBALD MCDONALD, M.D., Lecturer on Surgery, Nurses' Training School, Duluth, Minnesota. Pp. 265, with 46 figures. Philadelphia: J. B. Lippincott Co. 8s. 6d. net.

THIS book is an exposition of the principles of surgery explanatory of the anatomical and pathological facts underlying the nature and treatment of disease. It does not deal with operative technique. It is arranged in fourteen chapters, and a novel feature consists in suggested demonstrations at the end of each section, intended for practical classes. It ends with a short and rather incomplete glossary of terms.

Groundwork of Surgery. By ARTHUR COOKE, M.A., F.R.C.S., Surgeon to Addenbrooke's Hospital, Cambridge. Pp. 183. 1919. Cambridge: W. Heffer & Sons, Ltd. 7s. 6d. net.

THIS consists of the most elementary description of the first principles of surgery, and is intended to give first-year students a simple account of terms and principles, as an introduction to more serious text-books.

Reconstruction Therapy. By WILLIAM RUSH DUNTON, Jun., M.D., Junior Instructor in Psychiatry at the Johns Hopkins University. Large 8vo. Pp. 236, with 30 figures. 1919. Philadelphia and London: W. B. Saunders Co. 12s. 6d. net.

THE appearance of this book is very timely. It deals with the mental and physical training and occupation of the disabled, including orthopædic cases, mentally deficient, and the blind. It includes chapters on amusements, workshops, occupational training, prosthetic appliances, and gymnastics. It ends with a very useful list of reference books dealing in greater detail with the various branches of occupational training.

Prostatectomie sous Anæsthésie Locale. By PROFESSOR VICTOR PAUCHET, of Amiens. Pp. 25, with 20 illustrations. 1919. Paris: Maloine. 5 fr.

THIS small monograph records the experience of the author in 500 cases of prostatectomy. The mortality, which at first was 10 to 15 per cent, subsequently fell to 2 or 3 per cent, the last 50 cases being without a death. The improvement is attributed chiefly to conducting the operation in two stages, and to the use of local anæsthesia.

The diagrams illustrating the injection of the sacral plexus with novocain give the impression of a difficult procedure, and we think that equally good results might have been obtained by the use of gas and oxygen. Tamponade of the prostatic bed and section of the vasa deferentia in order to prevent septic orchitis are details of the method which are worthy of careful consideration.

Traitement Chirurgical des Affections de l'Estomac. By PROFESSOR VICTOR PAUCHET, of Amiens. Large quarto. Pp. 72, with 90 illustrations. 1919. Paris: Maloine. 12 fr.

THIS monograph takes the form of an atlas containing diagrammatic illustrations of the various operations practised by the author for gastric and duodenal ulcer and for gastric carcinoma. Of every 10 patients suffering from dyspepsia, about 9 are considered to owe their complaint to an extra-gastric lesion. The others are all cases of ulcer or cancer. Gastric cancer in 75 per cent is due to a pre-existing chronic ulcer. The pictorial representation of the operation is very clear and precise, but the letterpress is so scanty as to amount to little more than a short commentary on the pictures.

Report on the Medical and Surgical Developments of the War. By WILLIAM SEAMAN BAINBRIDGE, Lieut.-Commander, Medical Corps United States Naval Reserve Force. Large 8vo. Pp. viii + 250, illustrated. Government Printing Office, Washington.

THIS Report, published in January, 1919, forms a special number of the United States Naval Medical Bulletin, and as stated in the foreword, comprises observations on the western front and in England during December, 1917, and the first six months of 1918. The first section deals with the various methods of wound treatment and the administration of anæsthetics, and is followed by sections on such subjects as fractures, amputations, plastic surgery, and trench fever. Then follows a section dealing with the care of the wounded from the firing line to the convalescent camp, and consisting chiefly of notes made at visits to many hospitals in France and England, as well as military orthopædic hospitals and convalescent camps. The remainder of the report deals with, besides various miscellaneous matters, the re-education of the disabled and the principles of the treatment of wounds of war, chiefly as laid down by the Inter-Allied Surgical Conferences.

The Report undoubtedly contains a large amount of interesting information, but, especially in the section dealing with visits to different hospitals, much matter of no great value is introduced. The book is lavishly illustrated, but without affording a proportionate amount of interest or instruction to the reader.